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THE ARCHITECTURE OF THE BLOOD VASCULAR NETWORKS IN THE ERECTILE AND SECRETORY LINING OF THE NASAL PASSAGES.*

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The architecture of the networks in the mucous lining of the nasal passages will be discussed in connection with erection of the tissue and the concomitant production of the nasal discharge. An important part of the discussion of the angioarchitecture is a discussion of the presence of pseudo-arteriovenous anastomoses, or PAVAs, the absence of true arteriovenous anastomoses, or AVAs, and the probable reasons for the absence of AVAs in this erectile tissue. To a large extent the discussions will be limited, in this paper, to results obtained from some dead animals. Many animals were studied, but the figures showing the blood vessels in the nasal lining were obtained from the reindeer and the Walaroo kangaroo only. The reindeer was selected because its nasal lining is exceptionally vascular and erectile. The kangaroo was selected because its nasal lining is relatively avascular and is only slightly erectile. Aside from the figures showing the blood vessels in the nasal lining, two figures showing some AVAs are presented in order to indicate the merits of one of the methods used to determine the presence or absence of AVAs and to point out some reasons for their nonexistence in the erectile tissue of the nose.

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The photograph (Fig. 4) and all of the photomicrographs were obtained from the specimens after they were thoroughly dehydrated with absolute alcohol and cleared by the Spalteholz method of displacing the alcohol with benzene and then displacing the benzene with synthetic oil of wintergreen. Fig. 3 was obtained from the fresh, uncleared specimen. The Eastman infra-red plate 1R was used with the Wratten A filter in obtaining the infra-red photograph (Fig. 4) and three of the photomicrographs (Figs. 5, 6 and 7). The other infra-red photomicrographs were obtained by using the Eastman infra-red plate 1R without the Wratten A filter.

The arteries were injected first with India ink and then red cinnabar (Figs. 1, 2, 11, 12, 13 and 14) or with red cinnabar alone (Figs. 3, 4, 5, 6, 7, 8, 9 and 10). The cinnabar is radiopaque if it is packed firmly in the vessels (Fig 3). Upon using the 1R infra-red photographic plate with reflected light, the cinnabar photographs as brightnesses which range from white to neutral gray (Figs. 4, 6, 8, 9, 10 and 11). Upon using transmitted light instead, the cinnabar photographs as black (all of the vessels in Fig. 5 and all but the capillaries and some of the smallest arteries and arterioles in Fig. 14). The three-dimensional effect can be enhanced by superimposing the negative taken with reflected light upon the negative taken with transmitted light, rephotographing the superimposed negatives and then printing from the single negative (Fig. 7). The cinnabar can be photographed as a gray of a desired brightness by exposing a single infra-red plate to first reflected and then transmitted light (the relatively nontortuous vessels in Figs. 12 and 13). The cinnabar in the large arteries in the specimen presented as Fig. 12 (the large, relatively nontortuous vessels) photographed as light grays because the plate was exposed to both reflected and transmitted light but principally to reflected light. In the more highly magnified portion (Fig. 13) of the same specimen, the arteries are not entirely black but are much darker than those in Fig. 12 because the plate was exposed principally to transmitted light. Such variations in the photographic technic are useful for emphasizing different features of a single specimen. This is especially true when the arteries are injected with cinnabar and the capillaries alone or the capillaries, venules and veins are injected with India ink.

Upon injecting first India ink and then red cinnabar into the arteries, the ink is driven into some of the capillaries only (Fig. 14) or into the capillaries, venules and veins (Figs. 11, 12 and 13). If the cinnibar is not firmly packed in the arteries some of the ink may remain in some of the smallest arteries. The amount of the ink pass-

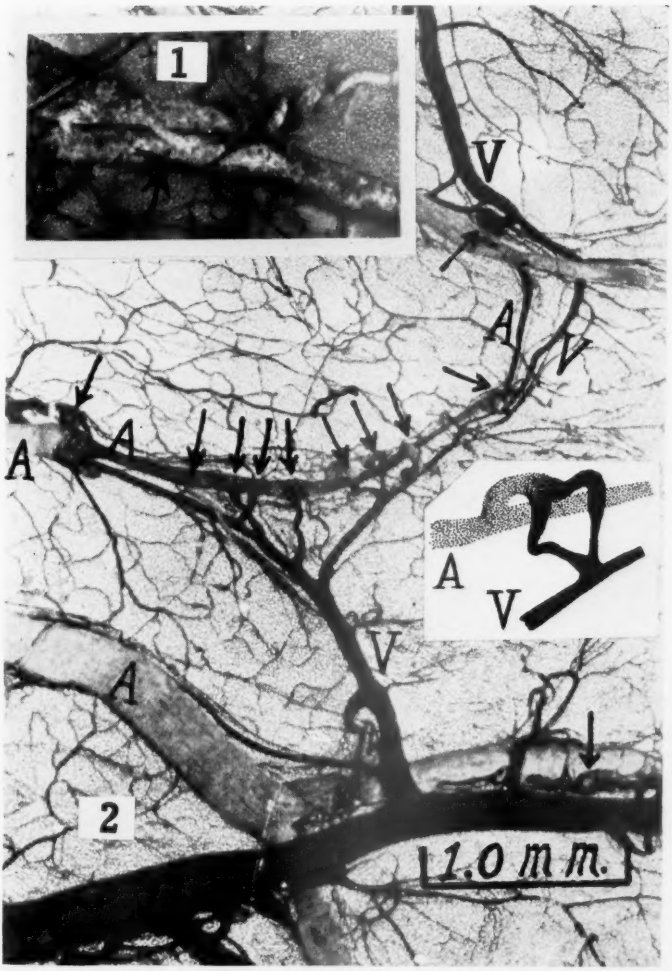

Fig. 1. Photomicrograph of two AVAs connecting an artery with a choked vein in the wall of the stomach of a five months' human fetus. The vein is the upper large vessel in the figure. Note the manner in which the vein and a branch of the large artery are coiled about one another somewhat peripherally to the anastomoses. The fetus was injected from an umbilical artery, first with India ink and then red cinnabar. The magnification is the same as that for Fig. 2.

Fig. 2. Photomicrograph of nine simple arteriovenous anastomoses (indicated by arrows) and one split one (indicated by two arrows and a bracket) in the ear of a rabbit. The split anastomosis is also represented with pen and ink. The arteries of the animal were injected first with India ink and then they were packed with red cinnabar. The skin was removed from both sides of the cartilage.

ing into and beyond the capillaries may depend upon the amount of ink injected as well as upon the amount of cinnabar packed in the arteries. Since the radiopacity of the cinnabar is poor unless it is packed firmly in the vessels, a roentgenogram of the fresh tissue can be used to determine whether the nonradiopaque ink has been driven entirely out of the arteries. If the roentgenogram shows that the smaller arteries are only slightly radiopaque, more cinnabar may be injected into and packed in the arteries until all but a certain irreducible minimum of the ink is forced out of them. For some purposes it is useful to leave some ink in the smaller arteries.

By using the infra-red plate and either transmitted or reflected light, ink in blood vessels photographs as grays ranging in brightness from a dark gray to black. The details, such as the capillaries, can be obtained more clearly with transmitted light only (Fig. 14) or transmitted light primarily (Fig. 13) than with reflected light primarily (Fig. 12) or reflected light only (Fig. 11).

Cinnabar can rarely be packed firmly enough in capillaries for these vessels to be visible in the photomicrographs. In the specimens presented as Figs. 7 and 8, for instance, injected capillaries can be seen, but they are not distinctly visible in the figures. The kind of light to which the infra-red plate is especially sensitive seems to make its way between and around either very loosely packed particles or very slender columns of cinnabar almost as well as it makes its way among the particles of ordinary fog. This kind of light is obstructed much better by the very dilute solutions of the nonradiopaque India ink. The very slender columns of India ink in the lumina of the capillaries can accordingly be demonstrated much better photographically than the very slender columns of cinnabar in the lumina of these vessels.

Under certain conditions an abundance of cinnabar can be urged through the capillaries and into the veins (Figs. 8, 9 and 10). This feat can be accomplished best by first diminishing the elasticity of the perivascular tissue while the intravascular fluid pressure is being altered in a rhythmical manner. The pulsatile variations in the vessels may be produced while the tissue is placed for several seconds in a moderately concentrated hydrochloric acid and then for an hour or more in tap water. A more convenient method consists in injecting a 2 or 3 per cent solution of hydrochloric acid into the arteries and varying the injection pressure for many minutes. The tissue becomes edematous and loses its elasticity to such an extent that if a portion of it is compressed with a blunt object, such as a finger, this portion remains deformed for some time. Likewise, when

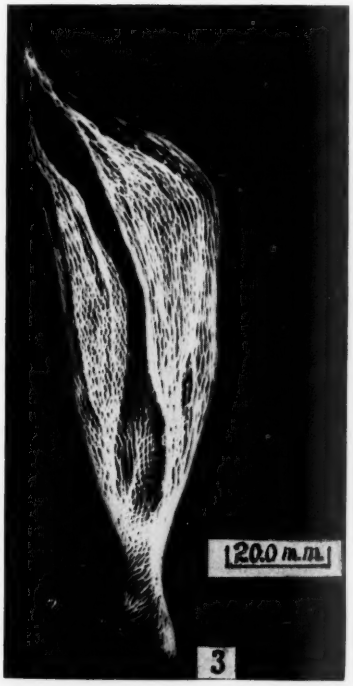

Fig. 3. Roentgenogram of a fresh ventral turbinate from the right nasal passage of a reindeer fawn. Red cinnabar was packed in the arteries.

Fig. 4. Photograph of the inferior turbinate of the fawn after the bone was decalcified and the tissues were dehydrated and cleared. Photographed with reflected light. Specimen immersed in methyl salicylate to avoid high lights.

the perivascular tissue is repeatedly forced aside by the walls of the rhythmically dilating vessels, it remains compressed for a sufficient length of time that the veins, as well as the arteries, can be packed with cinnabar by injecting this substance in a rhythmical manner into the arteries (Figs. 8, 9 and 10).

If no AVAs are present, cinnabar or India ink may be driven from the arteries into the capillaries but not entirely through them into the veins (Fig. 14) or the injected substance may be urged into the veins through the capillaries (Figs. 7-13). AVAs were not discovered in the mucous lining of the nasal passages of any of the mammals examined. Figs. 1 and 2 are presented in order to show that such pictures as Figs. 5, 6 and 14 could not be obtained from the mucous lining if AVAs were present in this lining. Instead of the

Fig. 5. Photomicrograph of a mounted portion of the cleared mucous lining of the right nasal passage of an adult reindeer. Red cinnabar was injected into the left common carotid artery. Photographed with transmitted light.

actual features of Fig. 14, the veins would have been inflated with India ink and cinnabar (as in Figs. 1 and 2) if AVAs had been present in the mucous lining. Figs. 1 and 2 indicate that it would be possible to locate the AVAs in the mucous lining of the nostrils if AVAs were present in this lining. Even the smallest AVAs which could be found in the ears of rabbits were injected in such a manner that they had a close resemblance to lighted neon tubes when these AVAs were examined with a binocular microscope and intense reflected light.

One of the AVAs in Fig. 2 has a large hole in it, i. e., a part of it has divided into two vessels. The two vessels of this AVA are pointed out in the photomicrograph by the two arrows under the brace, and a pen and ink reproduction of the entire AVA is placed to the right in the figure. This incompletely divided vessel represents

Fig. 6. Same as for Fig. 5 except that reflected light was used.

an early stage in the metamorphosis of an embryonic AVA into an arteriole and a venule connected with one another by a capillary network. As an embryonic AVA grows in length, it loops away from its original position and divides into two or more parts wherever the surrounding tissue causes opposite walls of the anastomosis to come in contact with one another. Some observations made principally on embryos of living viviparous snakes show that at the points of contact of the opposite walls, these walls fuse to form two or more blood channels out of one, and that the newly created vessels may separate widely from one another.

Each of the new vessels may loop away in different directions and divide into two or more parts and so on until the original anastomosis consists of a principal arterial trunk and a principal venous trunk which lie side by side and which are connected with one another at numerous points by relatively small arterial and venous branches and capillary networks. This metamorphosis may be of

Fig. 7. Composite photomicrograph of a mounted portion of the cleared mucous lining of the left nasal passage of the same adult reindeer from which Figs. 5 and 6 were obtained. A negative taken with reflected light was superimposed on a negative taken with transmitted light, the superimposed negatives were then photographed and a print was made from the single negative. The magnification is the same as for Figs. 5 and 6.

such a nature that the original AVA finally consists essentially of an arterial network, a venous network, numerous arteriocapillary anastomoses or terminal arterioles, numerous venocapillary anastomoses or terminal venules and numerous capillaries.

In some tissues of the body, as in and very near the cartilage of the pinna, some of the embryonic AVAs cannot loop away from their original positions. They are held in place by the solid tissue about them. These accordingly persist as relatively simple arteriovenous connections instead of very complex ones. The soft tissue of the mucous lining of the nasal passages is very much unlike the cartilage of the pinna. This soft tissue should not be expected to prevent the complete metamorphosis of the embryonic AVAs in it.

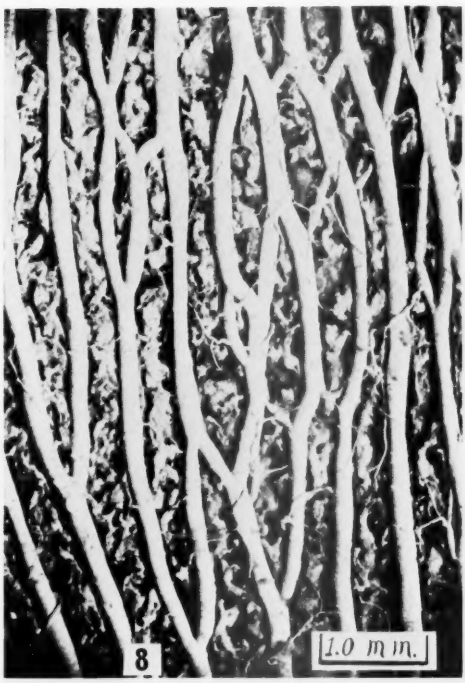

Fig. 8. Same as for Fig. 6 except that the specimen was from the left nasal passage of another adult reindeer. The head of this animal was injected first with a weak solution of hydrochloric acid to increase the lumina of the capillaries so that the cinnabar could be urged through them and into the veins.

Under certain conditions of congenital, partial obstruction of the flow of venous blood in a vein, embryonic AVAs fail to undergo metamorphosis at points somewhat centrally to the points of obstruction. Embryonic AVAs may accordingly persist in a tissue in which they are not ordinarily found. For instance, Fig. 1 shows two persisting AVAs in the wall of the stomach of a human fetus. These are situated somewhat centrally to the point at which an artery is choking the vein. The vein and artery are coiled about one another.

One may also observe that all of the AVAs in the nest of persisting AVAs in Fig. 2 are connected with two branches of a vein which pass underneath two arteries. Examination of the specimen shows that both of these arteries are partially imbedded in the carti-

Fig. 9. The same reindeer and the same nasal passage as for Fig. 8. This shows the deep veins as they may be seen through the decalcified and cleared bone or cartilage. The magnification is the same as for Fig. 8.

lage at the points where the venous branches pass under them and that the veins are compressed between the arteries and the cartilage. All of this nest of eight simple AVAs and one divided one can be said to be located centrally to the points of partial obstruction of the veins. The same is true of the AVA in the lower right corner of the figure, but the point of the venous obstruction is not shown in this photograph. The relatively dark slender portion of the artery which is associated with six simple AVAs and one divided one is completely imbedded in the cartilage. There are many other ways in which the flow of venous blood can be locally impeded to such an extent that the persistence of embryonic AVAs centrally to the points of the partial obstruction is favored.

The metamorphosis of an AVA into an arterial and a venous trunk and their branches implies growth. It is important to observe

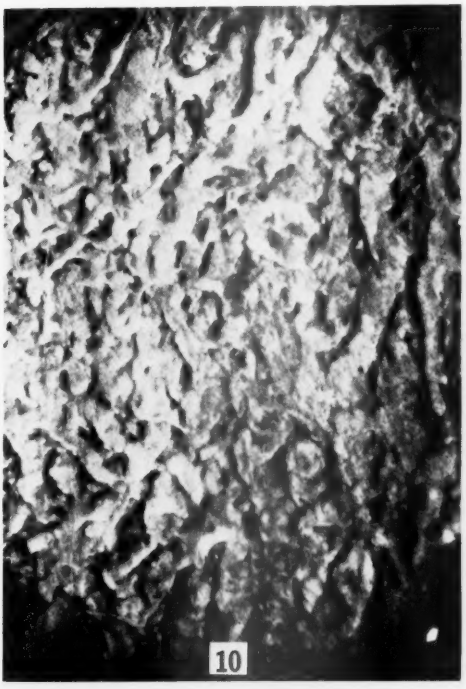

Fig. 10. An area showing a somewhat greater complexity of the venous network than is shown in Fig. 9. The magnification is the same as for Figs. 8 and 9.

in this connection that the growth and the resulting metamorphosis of the AVAs fail to occur at points where there is relatively little venous blood in the vein (due to the venous obstruction) and a relatively large amount of arterial blood in this vessel (due to the inflow through the AVA or AVAs from the artery) and that the portion of a leg peripherally to an arteriovenous fistula may grow more rapidly than the corresponding portion of the other leg (a well-known clinical fact). Such observations indicate that arterial blood in some way hinders growth and that venous blood in some way promotes it. The arteriovenous fistula in the leg shunts some arterial blood away from the distal portion of the leg, and the AVAs centrally to points of venous obstruction are exposed to arterial blood only or primarily, and accordingly fail to undergo the necessary growth for the metamorphosis of them. Perhaps the hydrogen ion concentration of the venous blood is the important growth factor.

If the network of veins in the walls of the stomach were as complex as the network in the mucous lining of a nasal passage, one or several partially occluded veins would have so little effect on the flow of the venous blood that the metamorphosis of the embryonic AVAs would not be arrested. It is true that in the postnatal mammal numerous veins are severely compressed every time the vein-like arteries become greatly dilated, but this compression takes place long after the embryonic AVAs have metamorphosed into the complex arteriovenous connections. The veins in this tissue are relatively simple vessels in embryonic and fetal life. The aneurismal dilatations and other irregular evaginated portions of the veins develop principally after birth. The veins become more complex with age. It is probable that they become more complex every time the tissue enters into a state of erection, as during a common cold.

The arteries in the lining of a nasal passage form a single network which will be spoken of somewhat abstractly as two interconnected ones. They are delicately interlaced at many points. Only one of these networks can be clearly seen in the roentgenogram (Fig. 3) and in the infra-red photograph (Fig. 4) of the same turbinate. The arteries of this network are extremely inflatable and are histologically very similar to many veins of the same size. These vein-like arteries have no branches which can be called arterio-capillary anastomoses, i. e., none of their branches are terminal arteries or arterioles. All of the vein-like arteries are arterial anastomoses. The other arterial network consists of relatively very slender arterial anastomoses which have a much greater resemblance to typical arteries. These anastomoses will be called typical arteries to distinguish them from the relatively large vein-like arteries. The typical arteries can be seen very clearly in Figs. 5, 6 and 14. They give rise to numerous arterio-capillary anastomoses which are the terminal arteries and arterioles.

Some of the small anastomotic connections between the vein-like arteries and the typical ones should be called pseudo-arterio-venous anastomoses or PAVAs because it is often impossible in the living tissue to distinguish between these and such true arteriovenous anastomoses or AVAs which are normal structures in the ear of the rabbit, according to Clark and Clark (1934) and others. In hundreds of instances anastomoses which seemed at first to be AVAs were proved to be only PAVAs. The problem of the presence of AVAs and PAVAs in this erectile tissue was given much consideration because Hoyer (1877) reported the presence of AVAs in the similar erectile tissue in the penis and because the notion is common at the present day that AVAs are undoubtedly present in such tissue.

Hoyer's observation could not be confirmed in this laboratory (unpublished comparative study by Dr. L. J. Deysach). The architecture of the blood vascular networks in the corpus cavernosum of the penis is fundamentally the same as that in the erectile tissue of the nose.

The vein-like arteries shown in Figs. 3 and 4 were greatly inflated with red cinnabar in water, and the mucous lining was accordingly in a state of exaggerated erection. About six weeks after the death of the fawn the arteries of the entire head were injected in a rhythmical manner from first one and then the other common carotid artery. The other arteries of the neck were occluded with ligatures and hemostats, but the veins of the neck were not closed. As soon as the animal died the head was removed from the body, drained of its blood, kept in a frozen state for about six weeks and then kept at ordinary room temperature for twenty-eight hours before the injection was made. The cinnabar was injected into the arteries in a pulsatile manner for a total time of about two hours in an effort to urge the cinnabar into all parts of the arterial, capillary and venous networks. However, the cinnabar particles could not be urged beyond the arterial capillaries at any point.

Figs. 5, 6 and 7 were obtained from the mucous lining of the nasal passages of an adult reindeer (the mother of the fawn). The animal was killed because of an irreparable condition of a leg. The head was quickly removed and perfused for about one minute with a 4 per cent solution of sodium citrate. About thirty-five minutes after the death of the animal, cinnabar in water was injected in a rhythmical manner into the left common carotid artery for a total time of about two hours. The other arteries of the neck were closed but all of the veins of the neck remained open. At all points to the right of the septal cartilage all of the arteries and many of the arteriocapillary anastomoses became well injected (Figs. 5 and 6). On the left (the side of the injection) a small amount of the cinnabar was successfully urged through some portions of the capillary network (Fig. 7). Some of the capillaries remained injected well enough to be seen clearly in the specimen, but these photographed very indistinctly. The cinnabar, which resembles snowdrifts between the arteries, is in the lumina of some irregularly distended veins such as can be seen much more clearly between the large arteries in Fig. 8 (from another reindeer) than in Fig. 7.

The lumina of the vein-like arteries to the right of the septum were increased about 200 per cent over the normal lumina in the living animal. The vein-like arteries to the left of the septum were

inflated still more. It is very difficult to estimate the increase over the bloodless condition of the arteries because many of them are almost flat when they are empty. The lumina of the typical arteries became only slightly greater when the cinnabar was packed in them. Even some of the typical arteries become almost flat when they are empty or almost empty. Although some particles of the cinnabar were urged through some of the capillaries on the left side, the arterial capillaries were sufficiently occluded everywhere by these stable emboli to permit an exaggerated state of erection to take place, as the arteries were inflated and packed with cinnabar.

It is probable that much of the injected water passed through the capillaries and into the veins, because the veins lying between the large arteries can be seen as greatly distended vessels in the fixed, cleared specimen. The water in the veins could not escape very readily because the venous avenues of escape were strongly compressed or mashed by the greatly inflated vein-like arteries. A considerable amount of water was trapped in the irregularly dilated veins which can be seen between the arteries of the specimen. These veins between the arteries will be called venous traps. They are venous water traps if only water is injected, venous water and cinnabar traps if water and cinnabar are injected together and they are venous blood traps in the living animal whenever the vein-like arteries become greatly dilated. The dilated vein-like arteries block the escape of fluid from these traps more than they block the flow of liquid into them through the capillaries.

As the arteries and venous traps became inflated and the erection was accordingly increased, a copious discharge streamed from the nostrils. This discharge was at first very slimy and viscous, but it eventually became almost the same as water. This change from the viscous to the watery type of discharge reminds one of the change in the nature of the discharge from the human nose in the course of a common cold. This is very commonly viscous at first and then watery, although the viscous, prodromic discharge from the relatively very small human nose is so small that it is commonly overlooked and is accordingly not ordinarily considered as being a part of the clinical picture. If the viscous, prodromal discharge is overlooked, the first discharge observed is a watery one, and in case of secondary infection of the mucous lining the watery discharge may be followed by a relatively slimy and viscous one.

The discharge from the nostrils of the reindeer was not colored with blood or cinnabar at any time, and no cinnabar could be recovered from the collected discharge.

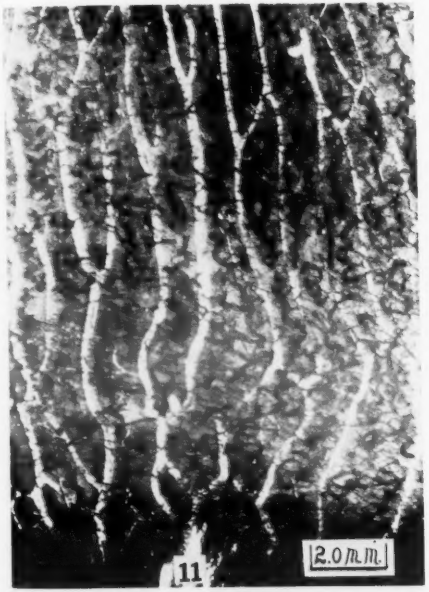

Fig. 11. Photomicrograph of a mounted portion of the mucous lining of the left nasal passage of a kangaroo. India ink was injected into the left common carotid artery and then this ink was driven into the capillaries and veins by injecting red cinnabar into the same artery. Photographed with reflected light.

Fig. 12. Same as for figure 11 except that both reflected and transmitted light were used instead of reflected light only.

Much of the viscous portion of the discharge was evidently the liquid which was already in the tissue outside the blood vessels before the injection was begun. Upon inflating the vessels each artery and each venous trap created a certain pressure (its fractionate pressure) upon the extravascular liquid, and the sum total of these pressures forced the liquid to escape into the nasal passages. The watery portion of the discharge continued as long as the injection continued, although it decreased slightly in amount as cinnabar was packed more and more firmly in the arteries. This liquid was evidently some of the injected water which seeped first through the walls of some blood vessels (principally the very thin walls of the venous traps) into the surrounding tissue and then into the nasal passages.

Upon injecting cinnabar in water under a constant high pressure into the arteries of other reindeer the viscous discharge occurred, but this diminished in amount without becoming watery until the injection pressure was varied in a pulsatile manner. Evidently, therefore, the viscous portion was already outside the blood vessels and was merely forced into the nasal passages when the arteries and venous traps were inflated. In order to explain the viscosity of it, the needed assumption is that during the life of the animal, when pulsatile variations were occurring in the intravascular blood pressure, some of the liquid of the blood seeped through the walls of some of the vessels and that especially the water of this filtrate was carried away by the air currents in the nasal passages. Only the more viscous part of the filtrate remained to be forced out of the tissue when the arteries and venous traps were strongly inflated after death.

When the injection pressure remained high and constant, the viscous discharge was not followed by a watery one because the perivascular tissue was so severely compressed that it was itself highly impermeable and that this compressed tissue was applied snugly enough against the vascular walls to prevent the outflow of water through any small openings in these walls. Upon altering the injection pressure in a rhythmical manner, the perivascular tissue and the walls of some of the blood vessels became alternately permeable and impermeable in a manner appropriate to urge water in a rhythmical manner into the nostrils.

It is unnecessary to attempt to explain any part of the nasal discharge of a dead animal in terms of a vital activity of the secretory cells in the membrane because similar results were obtained by injecting the head of the reindeer fawn after this head had been frozen for about six weeks and was then kept at ordinary room temperature for

Fig. 13. Same as for figure 12 except that the magnification is greater and that principally transmitted light was used.

Fig. 14. Photomicrograph of a mounted portion of the mucous lining from the right nasal passage of the same kangaroo. Photographed with transmitted light only. The magnification is the same as for figure 13.

twenty-eight hours. There was evidently no vital activity of the secretory cells in the fawn.

A similar viscous discharge followed by a watery one should be expected to occur during the life of a reindeer if a considerable number of the capillaries, a considerable number of the typical arteries or a considerable number of the arteriocapillary anastomoses in the nasal lining should become temporarily occluded by emboli or as a result of spastic contraction of these vessels. The discharge should follow the expansion of the vein-like arteries before as well as after death.

Figs. 8, 9 and 10 were obtained from an adult reindeer which was killed after it was severely gored by a black fallow buck. Immediately after the head was removed, thirty cubic centimeters of a 4 per cent solution of sodium citrate were injected into the blood vessels and then this liquid was permitted to escape from the cut arteries and veins of the neck. About thirty minutes later a 2 per cent solution of hydrochloric acid was injected in a rhythmical manner for about fifteen minutes into the left common carotid artery while the veins and the other arteries of the neck were occluded with hemostats. Cinnabar in water was then injected into the left common carotid artery for the same time and in the same manner as it was injected into the same artery of the adult reindeer from which Figs. 5, 6 and 7 were obtained.

Because of the loss of elasticity of the perivascular tissues (resulting from the injection of the acid) and because of the increase in the lumina of the capillaries (due to rhythmically distending the capillaries against the perivascular tissue while it was becoming inelastic), the cinnabar passed so readily through the capillaries that the venous traps became well distended with this substance (Figs. 8, 9 and 10). The cinnabar passed so readily into the veins that the vein-like arteries did not remain maximally distended after the injection ceased. Figs. 9 and 10 show some of the usual configurations of the deepest veins. The venous network is so dense that the relatively superficial vein-like arteries cannot be unmistakably identified by looking into the small spaces between the greatly inflated veins. The nasal discharge was essentially the same as that from the fawn and from the mother of the fawn.

Figs. 11, 12 and 13 were obtained from the left side, and Fig. 14 from the right side of the nasal septum of an adult Walaroo kangaroo (Macropus robustus, Gould). The cause of death could not be determined. Before the blood had had time to coagulate in the

vessels the head was removed and some India ink, diluted with a small amount of sodium citrate, was injected in a rhythmical manner, but very mildly, into the left common carotid artery. The other arteries of the neck were closed, but the veins of the neck remained open. Red cinnabar in water was then injected in a rhythmical manner into the same carotid artery until all of the arteries, which could be seen by dilating the nostrils, were well filled and distended with the cinnibar. Fig. 11 shows the vein-like arteries clearly, but the typical arteries contained such a mixture of ink and cinnabar that they were brown and accordingly photographed as grays which are indistinguishable from some of the grays of the background. This photomicrograph was obtained by using reflected light and the 1R infra-red plate.

In securing Fig. 12, first reflected and then transmitted light was used in order to emphasize some of the features of the vessels which contained India ink only or a mixture of India ink and red cinnabar. These features are emphasized still more in Fig. 13, which was obtained by using principally transmitted light. The capillary network is well injected almost everywhere. A comparison of the venous traps in Fig. 13 with those in Figs. 8, 9 and 10 shows that they are much simpler in the kangaroo than in the reindeer. The arterial meshwork in the kangaroo is much coarser and accordingly much simpler than that in the reindeer.

Fig. 14 was obtained from the left side of the nasal septum by using transmitted light only. In the absence of injected veins the typical arteries are unusually distinct in the photomicrograph. The vein-like arteries in this part of the lining are well filled with cinnabar, but some of the typical arteries are so mildly injected that they are flat and angular at some places in the cleared and shrunken specimen. The typical arteries probably never become flat in the living animal.

I wish to express my appreciation and thanks to Dr. L. J. Deysach, who regularly confirmed the principal observations herein reported. I am indebted to Mr. L. C. Massopust for the photographic technic and for the illustrations.

SUMMARY AND CONCLUSIONS.

1. The arterial network in the mucous lining of a nasal passage of a postnatal mammal consists of two interconnected and intricately interlaced networks. One of these consists of numerous, thin-walled, very inflatable, vein-like arteries which do not give rise to arteriocapillary anastomoses. The other network consists of numerous slen-

der vessels which are more like typical arteries of the same size elsewhere in the body. The typical arteries give rise to numerous arteriocapillary anastomoses. Some of the connections between the two networks resemble true arteriovenous anastomoses (AVAs), and were accordingly called pseudo-arteriovenous anastomoses (PAVAs). AVAs could not be found in the erectile tissue of the nose. The arterial networks are much simpler in the kangaroo than in the reindeer.

2. The network of veins is extremely complex in the reindeer but relatively simple in the kangaroo. Numerous veins in both animals are very greatly and irregularly dilated.

3. Upon inflating the vein-like arteries with a liquid, these arteries compress veins that pass underneath and above them so that a considerable amount of liquid is trapped in the irregularly dilated veins in the spaces between the vein-like arteries. These veins were called venous traps for blood, water, cinnabar, India ink or any other substance which might be used to inflate the vein-like arteries and incidentally the venous traps.

4. Upon inflating the vein-like arteries and the venous traps to cause erection of the mucous lining, liquid which is already in the perivascular tissue is driven into the nasal passages as a viscous discharge. If the intravascular pressure is varied in a rhythmical manner the viscous discharge is followed by a watery one, as water is filtered through the walls of some blood vessels and then urged into the nasal passages.

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XCVI.

CANCER OF THE EPIGLOTTIS: TOTAL EXTIRPATION OF THE EPIGLOTTIS BY THE LARYNGOFISSURE ROUTE.*

GABRIEL TUCKER, M. D.,

PHILADELPHIA.

Cancer invading the epiglottis is ordinarily classified in the extrinsic group of laryngeal malignancies. Extrinsic malignancy as a rule gives rise to early metastasis and extension of the growth. In cancer of the epiglottis this holds true when the cancer invades the upper or lingual surface of the epiglottis. Cancer of the under surface of the epiglottis, however, may be slow growing and very slow to metastasize and in this regard it resembles more closely the intrinsic cancers probably due to the barrier afforded by the cartilage of the epiglottis. In cancer of the under surface of the epiglottis conservative surgical treatment may effect a cure although the lesion has existed for a considerable length of time. If the disease has not penetrated the cartilage and there is no evidence of metastasis, removal of the epiglottis should be sufficient to effect a cure.

Methods of Removal of the Epiglottis.—Three methods of treating cancer of the epiglottis are in common use. 1. Removal of the epiglottis by thermocautery or snare through the mouth by means of suspension laryngoscopy. 2. Excision of the epiglottis by the transhyoid approach. 3. Excision of the epiglottis by the lateral pharyngotomy approach. When the growth extends downward involving the ventricular bands the procedure most commonly employed is total laryngectomy.

A case presented itself in which the growth was limited to the under surface of the epiglottis at and below the point of attachment to the hyoid bone, rendering the case unsuitable for excision of the epiglottis through the mouth. After study of the case it seemed logical to approach the epiglottis by a laryngofissure incision. In this way the interior of the larynx could be carefully inspected in order to determine the exact limit of the extent of the growth downward

*Presented before the annual meeting of the American Laryngological Association, Toronto, May 29, 1935.

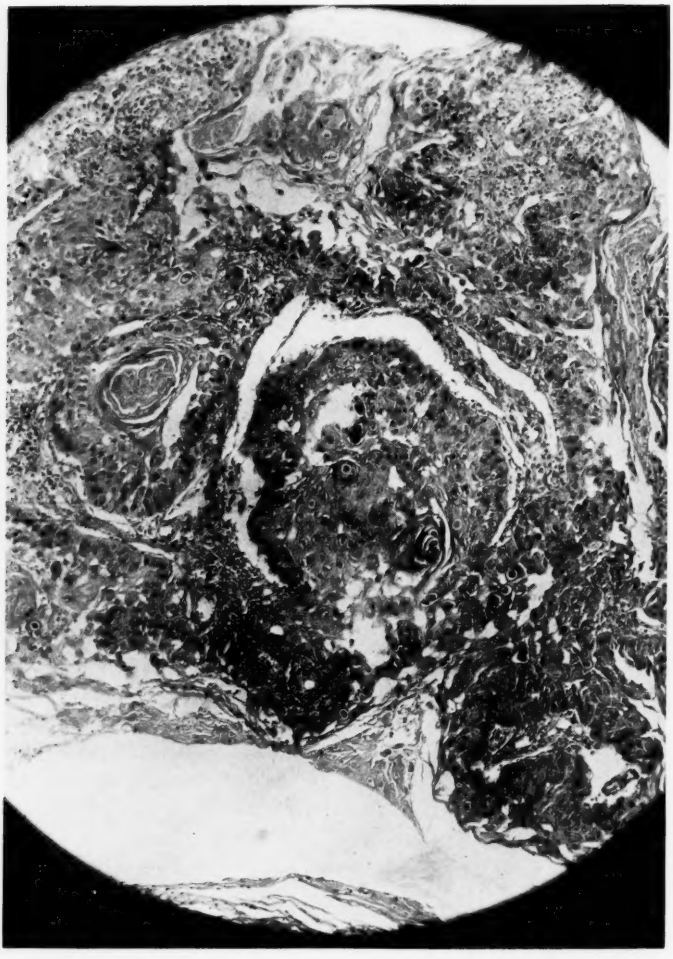

Fig. 1. Microphotograph of tissue removed from the under surface of the epiglottis by direct laryngoscopy, showing "carcinoma grade No. 1." (Dr. E. A. Case.)

and by incising the thyrohyoid membrane upward to the level of the hyoid bone adequate exposure of the base of the epiglottis could be obtained to allow total removal. Total extirpation of the epiglottis was therefore carried out by the laryngofissure route.

REPORT OF A CASE.

CASE NO. C.S.G. 180. Male, aged sixty-seven years, by occupatian, clerk. Had complained of local discomfort in his throat for the past year. This was accompanied by pain radiating to the left ear when he attempted to swallow. Never continuously hoarse. Family history was negative. Moderate user of tobacco, no alcohol. General examination was negative. Patient had an osteochondroma of the maxilla which, on consultation with Dr. George Coates, we were assured had no bearing on his laryngeal condition and was not a contraindication to the operation on the larynx. Mirror examination showed the under surface of the epiglottis presenting an ulcerating area extending from the midline toward the left fairly well down to the ventricular band on the left side. The surface was elevated and had the appearance of a cancerous infiltration. The lesion did not extend to the margin of the epiglottis and there was no involvement of the upper surface of the epiglottis. Careful examination of the neck showed no adenopathy. Direct laryngoscopy and biopsy confirmed the mirror findings and the tissue submitted to Dr. E. A. Case showed squamous cell carcinoma, Type No. 1. (Fig. 1.) The decision to treat the patient by removal of the epiglottis only was discussed with Dr. John L. Johnson, the referring physician, and the patient came in for operation. Lateral roentgen-ray examination of the neck demonstrated well the lesion on the under surface of the epiglottis. (Fig. 2.)

Realizing that several difficulties might be encountered during and after the removal of the epiglottis we did a preliminary tracheotomy as the first stage. We hoped that preliminary tracheotomy would aid in the control of hemorrhage should it occur and also help to prevent aspiration of food into the tracheobronchial tree during the time that the patient was acquiring the proper compensatory protective mechanism after the removal of the epiglottis.

Tracheotomy under local anesthesia was done December 31, 1932. The tube was placed low. A transverse incision was made just above the suprasternal notch, a small section of one tracheal ring was removed and a No. 5 tracheotomy tube was inserted. Ten days later the epiglottis was removed.

Thyrotomy for Removal of the Epiglottis.—Anesthetic, avertin 70 mg. per rectum, novocain infiltration. Operation: Midline vertical incision from the hyoid bone to a point just above the tracheotomy fistula. The front of the larynx was exposed from the cricoid cartilage to the thyroid notch. Cricothyroid membrane was incised vertically, thyroid cartilage divided with a turbinotome. The wings of the thyroid cartilage were retracted and an excellent view of the interior of the larynx showed the lesion to be limited to the under surface of the epiglottis and to extend downward to a point about 1 cm. above the ventricular band. The involvement was on the left half of the epiglottis on its under surface. In order to be well beyond the growth a subperichondrial resection of the inner surface of the

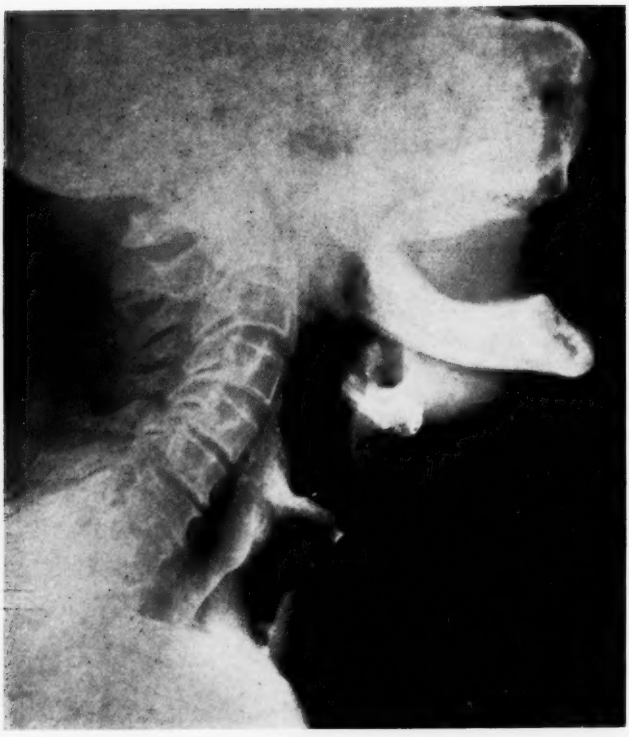

Fig. 2. Lateral roentgenogram of the neck showing location of the lesion on the under surface of the epiglottis. (Roentgenogram by Dr. Pancoast.)

left wing of the thyroid for about 1 cm. from the left side of the thyroid notch was done. This excision included the anterior end of the left ventricular band. The same procedure was carried out on the right side. Hemorrhage was controlled without difficulty. The thyrohyoid membrane was divided sufficiently to allow exposure of the base of the epiglottis. The base of the epiglottis was grasped and pulled downward and the mucous membrane on the upper surface of the epiglottis was removed by subperichondrial dissection. This freed the epiglottis except at its attachments on either side. The incisions through the ventricular bands were extended upward; the tissues removed, including the aryepiglottic fold on either side, and the epiglottis was removed. (Fig. 3.) The bleeding points were easily grasped as a good exposure was obtained. Points were ligated with suture ligatures and the flap of mucous membrane which had pre-

Fig. 3. Photograph of the epiglottis after removal. The arrows indicate location of cancer on the left side of the under surface of the epiglottis.

viously covered the upper surface of the epiglottis was pulled downward, covering the denuded area on the front of the hyoid bone where the base of the epiglottis had been previously attached. A small piece of cartilage was removed from the upper corner of the thyroid wings on either side, the external perichondrium being dissected off from the removed cartilage and left in position. This permitted the thyrohyoid membrane and the tissues on either side to be pulled together with a deep silkworm gut suture, which was carried around from the skin surface on one side, encircling the tissues where the base of the epiglottis had been attached and coming from within outward through the skin on the opposite side. This deep suture closed completely the opening remaining after the removal of the epiglottis. The laryngofissure wound was closed after the usual technic, using interrupted silkworm gut sutures.

No difficulty was encountered in removing the epiglottis by this method. The excision was entirely above the level of the true cords, and mirror examination following the removal showed that the cords approximated perfectly and that their function had been disturbed very little.

Postoperative Course.—The convalescence was uneventful. A feeding tube was placed through the nasopharynx immediately fol-

lowing the operation and all fluids were given by tube. The only difficulty encountered was after the fifth day when we attempted to remove the feeding tube. The patient complained of great difficulty and choking when he attempted to swallow, although there was very little food passed beyond the level of the cords. He gradually overcame this difficulty, however, and at the end of the seventh day the feeding tube was removed. The patient left the hospital at the end of two weeks, the tracheotomy tube having been removed and the difficulty in swallowing having been practically overcome.

I have had frequent reports on the progress of this patient, but have not had the opportunity of seeing him. In January last, at the meeting of the Section of the Triological in Portland, Maine, Dr. Louis H. Clerf very kindly saw him for me and reports that there is no evidence of recurrence and that his general condition is excellent. The report six months later, now three and a half years following the removal of the epiglottis, from Dr. John L. Johnson, the laryngologist who originally referred him, states that his larynx is in excellent condition.

COMMENT.

1. A case of cancer of the under surface of the epiglottis is presented in which total extirpation of the epiglottis was done by the laryngofissure route was reported as cured three and a half years following the removal of the epiglottis. The larynx functions normally, including a normal voice.

2. Attention is called to a type of cancer of the epiglottis that is slow growing and late in extension beyond the epiglottis. A method of conservative surgical treatment is suggested by total extirpation of the epiglottis by the laryngofissure route.

326 S. 19TH ST.

XCVII.

ANATOMIC ANOMALIES OF IMPORTANCE TO THE OTOLARYNGOLOGIST.*

OSCAR V. BATSON, M. D.,

PHILADELPHIA.

The subject of anomalies is always intriguing. Perhaps it is their seeming mysterious character that appeals. We have the feeling that here is something beyond our power of comprehension. An anomaly should, however, challenge us to interpret the structure which has been defined as "contrary to rule." I have always been impressed with the extremely normal appearance of anomalous structures as they lie in the body. The whole organism is adapted to them. It was this normal appearance, coupled with the small number of specimens available to him, that led Vesalius to describe as normal an anomalous ossicle in the foot, the os Vesalii.

Since the otolaryngologist looks upon each skull in a collection as an unusual specimen, especially in reference to the paranasal sinuses, to even enumerate all of the observed anomalies of interest would be impossible. To narrow the field and to allow for a more detailed consideration, I have chosen to describe some of the vagaries of the arterial system that are found in the head and neck. These are of considerable practical importance. Some of these anomalies are due to abberations of development, and as such their presence may not be forecast. When encountered, however, it may be possible to interpret their presence.

One of the most unusual of such anomalies is the doubling of the aorta. Such a case was described by Dr. Homer Blincoe et al. (1932). In this specimen, which I have had the opportunity to examine, the aorta splits to enclose the trachea and esophagus and recombines posteriorly. The genesis of this condition, with a review of the older literature, will be reported by Dr. Blincoe. This is a very rare anomaly, but not unique. While it probably would not occlude the trachea and esophagus, it would give peculiar radiographic and endoscopic findings.

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*Paper read before the forty-first annual meeting of the American Laryngological, Rhinological and Otological Society in Toronto, Ontario, on June 3, 1935.

There is an anomaly, however, due to peculiarities of transformation of the aortic arches, which is of decided interest to the otolaryngologist. It occasionally happens* that the right subclavian artery does not spring from the innominate artery, but comes from the descending part of the thoracic aorta. Under these circumstances, due to the disappearance of the proximal portion of the embryonic right aortic arch, the right recurrent laryngeal nerve is not displaced inferiorly by the right subclavian artery. It comes directly from the main stem of the vagus nerve and courses directly over to the larynx posterior to the superior thyroid vessels. Considering that the upper thyroid pole may not be handled with as great care as the tracheoesophageal angle, it is obvious that this "recurrent" laryngeal nerve may be damaged in the thyroid operation.

There are a number of other anomalies which, like the two major ones just noted, would fall under the head of irregularities of transformation of the embryologic vascular pattern. To be included here would be the origin of the external maxillary artery from the internal maxillary artery and its consequent relation to the parotid gland instead of its usual relation to the submaxillary gland.

Mention should also be made of such balancing arrangements as are commonly seen. The transverse scapular and the transverse cervical arteries are of reciprocal size. This is true also of the anterior and posterior ethmoid arteries. When the surgeon finds a small anterior ethmoid artery in the radical fronto-ethmoid sinus operation, he should expect this to be compensated by a large posterior ethmoid artery.

There are a number of anomalies of the arterial system that have been described as anomalies of position, which upon more complete study may be shown to be fundamentally anomalies of size, i. e., length and diameter. One of the most interesting is to be found in the anomalous innominate artery. Dr. Chevalier Jackson (1934) has called attention to the relative safety for surgical approach to the trachea of a midline triangular area lying medial to the two sternocleido-mastoid muscles. So safe is this triangular area for tracheotomy that Dr. Jackson has labeled it "safety." However, in the specimen here described the innominate artery appears in the neck on the

*The frequency of occurrence of this condition depends upon the racial background of the material examined. Adachi (1928) has furnished some tabulations on this point. In an English series it was seen five times in 500 cases. Adachi saw but one case in 516 cadavera (Japanese) examined. In those American laboratories reporting the findings in the negro, the incidence is much higher. At Pennsylvania we see one or two a year.

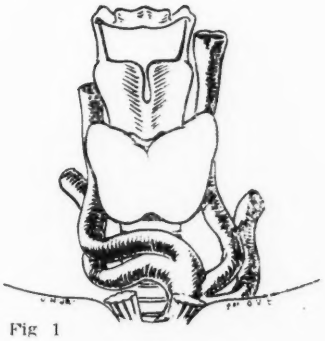

Fig. 1

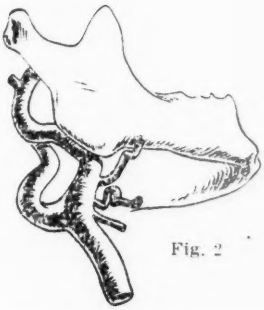

Fig. 2

Fig. 3

Fig. 1. Innominate artery with its branches lying in front of the trachea in supersternal area. Adult, black, male.

Fig. 2. Tortuous artery at the angle of the jaw in cadaver with extensive dilation and tortuosity of all vessels. Adult, black, male.

Fig. 3. Diagram of principal vessels of head and neck displaced by arteriosclerosis and hypertension. The arrow indicates that portion of the artery causing erosion of the carotid canal.

left side of the trachea, crosses the trachea in the triangle of safety to divide near the right border of the trachea into the left subclavian artery and the left common carotid artery (Fig. 1). This indeed would seem to be an anomaly of position and, of course, it is. However, an examination of the other large vessels shows, first of all, that the first part of the aorta is markedly dilated and lengthened. It presents the typical picture of the aorta dilated by hypertension and arteriosclerosis. This lengthening of the first part of the aorta caused some torsion of the heart and at the same time pushed the point of origin of all the vessels to a greater distance from the heart.

Here then we have an apparent anomaly—it has been described as such*—which, on analysis, is seen to be due to a pathologic change in the vessel. The anomaly of position is coincidental or a result of the pathologic change. The importance of this vessel to the operation of tracheotomy is at once apparent. Palpation of the triangle of safety would prevent surprise. This condition is seen at least once every other year at the laboratory of the Graduate School of Medicine. This means that it occurs about once in 200 cases in our material. It seems to be more common in the negro.

Another vessel, sometimes seen apparently out of its normal course and which obstructs a surgical approach, is the inferior thyroid artery. The retropharyngeal space and posterior mediastinum may be approached low in the neck. This site has been used for operation on cervical esophagus. Normally an incision through the skin and superficial fascia allows an approach to the area. The great neck vessels are retracted forward. In two or three out of each 100 of our specimens, the inferior thyroid bends cranialward as an inverted "U" (Fig. 4). When seen through the operative field, the vessel at first causes confusion. It is so large and so far from the usual course of a vessel. Complete dissection, as is possible in an anatomic laboratory, shows this to be a dilated, elongated, inferior thyroid artery bending upward and into the areolar tissue of the retropharyngeal space. It springs, by way of a rather tortuous thyroid axis, from a twisted and dilated subclavian artery showing the character of hypertensive arteriosclerotic vascular disease. This vessel seems to be more commonly displaced on the right side of the body.

The common carotid arteries and their branches also show anomalies due to increase in diameter and length. This may vary from a slight sinuosity to the formation of a complete loop. The complete loop, when present, is seen in the course of the internal carotid artery. The feature of interest to the otolaryngologist is the appearance of these vessels when exposed by the oral approach. This elongation of the arterial tree no doubt explains many, if not most, of the "anoma-

*Symington (1887) described this condition as normal for the child. His report seems to be the fundamental basis for the acceptance of this relation as normal by Merkel (1899) and others. Personally I have never found the innominate artery about the sternum in the newborn. I have not had the material available to investigate this point in children. In the adult specimens that I have seen, this condition is always associated with gross pathologic changes in the arteries. The case illustrated by Adachi (1928, p. 27) appears to fall into this class of pathologic vessels. The figure of the case reported by Jaensch (1922) undoubtedly represents a case of arteriosclerosis. This is also the opinion of Adachi (1928, p. 43) concerning the case of Jaensch.

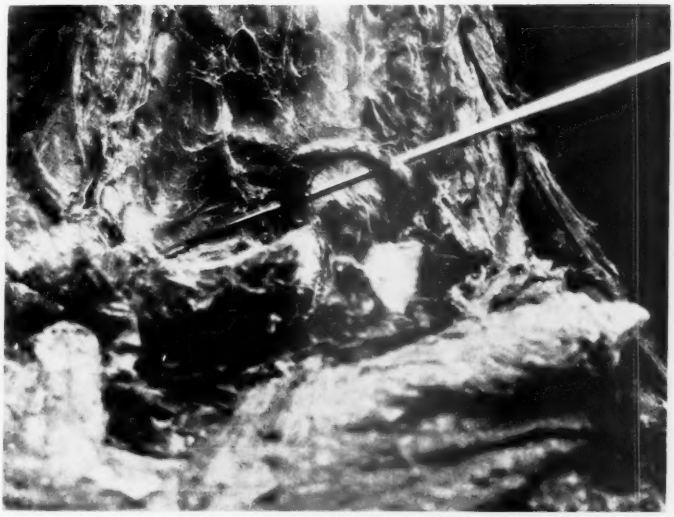

Fig. 4. Inferior thyroid artery extending upward in an inverted "U".

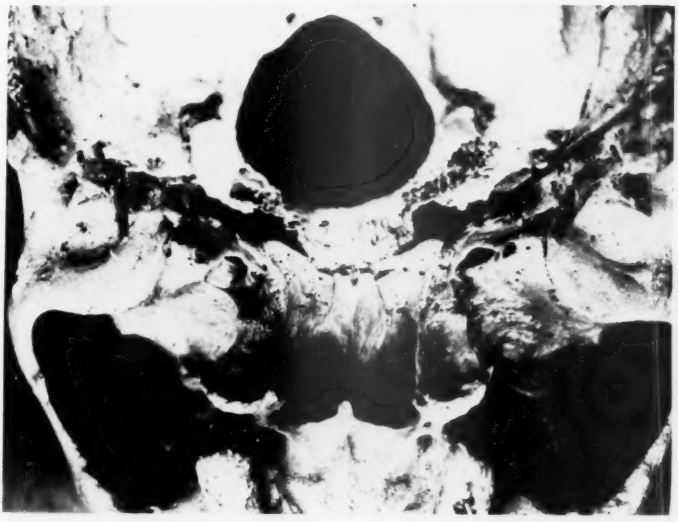

Fig. 5. Carotid canals converted into carotid grooves by enlargement of the internal carotid artery. This view indicates the relation in the mento-vertical roentgenogram.

lous" vessels seen in adults during tonsillectomy. Occasionally such vessels are seen in children and they may be due to a genuine congenital anomaly. I am inclined to feel that most of the adult cases are due to vascular disease. The purported tortuous carotid artery of the seal has been mentioned to provide phylogenetic reference for this condition. Carmel (1928) has shown that this observation of Chauveau has not been confirmed by other authors,* and he himself could not confirm it on twelve dissections of the seal. In man the artery need not make a complete loop to obtrude into the retrotonsillar area (Fig. 2).

The next vascular peculiarity to be examined is the carotid artery in its course through the bony carotid canal. Here a dilation with some increase in length causes an erosion of the carotid canal. This is significant because of a possible pitfall in diagnosis. With the recent interest in erosion of the apex of the petrous portion of the temporal bone through infection, many x-ray examinations are being made of this area. In the vertico-occipital view of this region, the erosion caused by the dilating carotid may be so closely related as to confuse the uninitiated. The mentovertical view differentiates the two areas clearly and should be more useful here (Fig. 5). Should there be, in addition to an eroded carotid canal, a petrous apex infection, it should be remembered that this enlarged artery makes smaller an already tiny operative field.

Unfortunately the many turns of the internal carotid artery have not been named. This makes the description of abnormal turns difficult. The normal carotid does not lie above the floor of the sella turcica when the skull is viewed in roentgenograms, or in the anatomic specimen. Under the condition of increase in length under discussion, this is not true. These extensions toward the vertex, when partially calcified, are seen by the roentgenologist. The otolaryngologist finds interest in the specimens in which the bend in the internal carotid artery, between the foramen lacerum and the side of the

*The first French edition of Chauveau (1857) and the second edition of Chauveau and Arlong (1873) were not available to Doctor Carmel. I have examined these and find no reference to the miraculous artery of the seal. Fleming (1871) introduced this statement into the first English edition of Chauveau and it has appeared in subsequent editions.

Smith (1902) in reporting his human case refers to the tortuosity of all the vessels. Fisher (1915) and Schaeffer (1921) inclined to the phylogenetic explanation. The more complete the exposure of all of the great vessels at one time, the more obvious the generalized nature of the condition became. Adachi (1928, p. 98) notes: "Da die Tortuosity mir von keiner morphologischen Bedeutung, sondern eine Alterserscheinung zu sein scheint, habe ich sie nicht statistisch untersucht."

Fig. 6. Probe passing through dehiscence (posterior) in enlarged carotid impression. Seen from cranial cavity.

Fig. 7. Probe passing through dehiscence (anterior) in enlarged carotid impression.

sella, has so enlarged medially that the bony wall of the sphenoid sinus has been eroded (Fig. 6). This erosion is in direct line from the pyriform orifice through the sphenoid ostium. Anteriorly the same dehiscence may occur (Fig. 7). Here the loop of the artery, as it bends forward and upward from the sella, has exposed the sinus. The clinical significance of these exposed vessels is obvious. The difficulty of making the differential diagnosis between tumor and enlarged carotid artery will not be discussed.

The sphenoid sinus is crossed anteriorly by the sphenopalatine artery. I am aware that Canuyt and Terracol (1925)* minimize this vessel as a source of severe hemorrhage. However, I have frequently seen what appears to be an abnormal, large, displaced vessel. This, when examined in the light of our discussion, is seen to be a pathologic condition involving many vessels rather than an anatomic anomaly. The large vessel sometimes encountered on the floor of the nose may fall into the same category.

SUMMARY.

1. There are many anomalies of the vascular system of interest to the otolaryngologist; some of them, the true anomalies, are not readily forecast.

2. A large group, which we might call pseudo-anomalies, are here shown to be due to pathologic changes in the vascular system, and their presence may be suspected in cases of peripheral arteriosclerosis, especially when associated with hypertension.

3. A diagram of these pseudo-anomalous vessels here discussed is presented as if found in one individual (Fig. 3).

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XCVIII.

THE ASSOCIATION OF FILTRABLE VIRUS AND BACTERIA IN THE PRODUCTION OF EXPERIMENTAL SINUSITIS.*

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ST. LOUIS.

There is no general agreement on the mechanism of the process resulting in purulent sinusitis in man. However, the trend of recent investigative work suggests that the tissues are prepared by a filtrable virus, which is then followed by bacterial invasion. A great majority of cases are sequelæ to the common cold or influenza. The uncomplicated common cold or influenza are mild diseases, but when accompanied by bacterial invasion the results may be serious. Dochez, Shibley and Mills[1] have demonstrated the presence of a filtrable virus in the common cold which is capable of producing symptoms of the disease in apes. A similar but somewhat different virus was found in cases of influenza by Smith, Wilson, Andrews and Laidlaw.[2] Shope[3] had previously discovered a filtrable virus in swine influenza and Dunkin and Laidlaw[4] in dog distemper, diseases which resemble human influenza in many respects. Snuffles in rabbits is a spontaneous infection of the sinuses and upper respiratory mucosa which frequently results in complications and death. Pneumococci, streptococci, staphylococci, influenza bacilli and other bacteria associated with sinusitis in the human are frequently present in the apparently normal nose and throat, and their virulence might be activated by a virus infection.

A study of the various methods of treating purulent sinusitis by nonoperative procedures has been retarded by the lack of a suitable experimental animal. The rabbit suffers from spontaneous sinusitis in which Pastuerella lepisepticum is found in large numbers. These organisms may be found in normal rabbit sinuses in a high percentage of cases.

Diseases of the respiratory tract, of virus or probable virus etiology, show a recognized predisposition to bacterial invasion. McCor-

*From Oscar Johnson Institute, Dept. of Otolaryngology, Washington University School of Medicine. (This work was made possible by the Ball grant.)

dock and Muckenfuss[5] produced in rabbits a typical picture of interstitial bronchopneumonia, a disease which is found associated with influenza, measles and whooping cough in man. They used dilute vaccine virus which was followed by injection of bacteria. The virus alone did not produce the typical picture. They were able, by using larger quantities of virus and bacteria, to simulate the hemorrhagic bronchopneumonia found in epidemic influenza. The pneumonias associated with dog distemper and swine influenza show similar pathologic pictures.

Various methods of producing sinus infections in animals have been used but none of them gives an infection which can be considered as analogous to the process taking place in a normally infected sinus. Preliminary traumatism of various kinds has been the usual mode of procedure. The amount of work of this kind is surprisingly small. Fenton,[6] 1931, injected bacteria alone, bacteria following dull curettage, and bacteria following heat, into the frontal sinuses of cats in a study of the "reticulo-endothelial" components of the sinus mucosa. Warm paraffin was used as a source of heat to plug the nasofrontal duct. The cellular changes with the various procedures are described. He reports that "bacterial inoculation after traumatism produced only an intensification of symptoms over bacteria alone." Fenton,[7] 1934, also reports producing unilateral frontal sinusitis in cats, using hemolytic streptococci. The exact technic is not given in this paper, but the infection apparently became chronic in at least some of the animals. Wenner and Nemours[8] produced an accumulation of eosinophilic cells, and in addition chronic infection in some cases, by repeated injection of sodium alizarin sulphonate solution into the maxillary sinuses of rabbits.

The attempt to produce sinusitis in animals by the injection of bacteria alone has resulted, in the hands of most workers, in the production of an uncontrolled fatal infection, only a temporary transitory effect, or, more commonly, no infection at all. Linton,[9] using an extremely virulent strain of hemolytic streptococcus, was able to produce rapidly fatal infection in guinea pigs and rabbits by simply dropping some broth culture into the nose. The production in animals of sinus infections, suitable for experimental studies, has, in most instances, therefore, been quite unsatisfactory. When it was desired to take up a problem requiring an established purulent sinusitis a satisfactory condition was found difficult to secure. The rabbit was selected because of the ease with which the maxillary sinuses may be reached. It has the disadvantage of being subject to spon-

taneous nasal and sinus infections. This factor may be largely eliminated by proper methods and controls.

METHODS.

The requirements of a satisfactory sinusitis in rabbits for experimental purposes are that: (1) it should be produced with a fair degree of regularity; (2) it should be sufficiently chronic to allow passing of judgment on the results of treatment methods, and, (3) it should resemble as much as possible a natural sinus infection. These requirements have to a great extent been fulfilled.

Since a virus seems to be involved in the beginning of a high percentage of human sinus infections, the use of a virus in the production of the experimental disease naturally suggests itself. Vaccine virus was selected of a type used in vaccinating for smallpox. The rabbit is susceptible to this virus, and the quantity used was one to two times the amount used for vaccination. The bacterium used most extensively was a hemolytic staphylococcus aureus isolated from an infected tonsil. Other bacteria used were a mixture of strains of Streptococcus hemolyticus, H. influenza, Bacterium lepisepticum, and in one case each, Ps. pyocyaneus, Str. viridans and the colon bacillus. The rabbits were healthy adult animals and usually white, because nasal discharge is easily detected with this color.

A preliminary experiment indicated that there was no advantage in making the virus injection twenty-four hours or more before injection of the bacteria. The usual procedure, therefore, was to draw the virus through a small needle into the syringe and then draw the bacterial suspension into the same syringe, thus mixing thoroughly the virus with the bacteria. The mixture was then injected through the lateral wall of the nose into the right maxillary sinus of the rabbit. The left sinus was injected with a similar amount of physiologic saline as a control. The bacterial suspension was made up in most instances by washing the growth from a twenty-four hour blood agar plate into five cc. of saline. One and four day whole broth cultures, and one and four day supernatent fluids from broth cultures were tried.

The virus was usually injected only once, but bacterial suspensions were injected one to four times, usually three times, at varying intervals. The best method was to inject the virus-bacteria mixture on the first day and the bacteria alone on the two following days. The hair was clipped closely over the regions through which the injections were made. The animals were observed daily until autopsied, for nasal discharge or other signs of illness.

Duran-Reynolds,[10] 1929, described the enhancing effect of testicular extract on vaccine virus injected into the skin of rabbits. He prepared his own testicular virus and injected normal testicular extract along with it. Rabbits with enhanced lesions showed general symptoms and 25 per cent died of vaccinia. The virus could be inactivated by immune testicular tissue. The same author,[11] 1933, found that invasive strains of staphylococci and streptococci contain a soluble factor greatly increasing tissue permeability and enhancing infections. Noninvasive strains did not contain this factor.

Hoffman,[12] 1931, observed that testicular extract enhances the virulence of the viruses of herpes, vesicular stomatitis, vaccinia and Borna's disease. Pijoan,[13] 1931, found that it promotes the pathogenic action of twenty kinds of ordinary bacteria. Hanger,[14] 1931, showed a similar phenomenon, using nasal secretions from normal persons and persons with common colds, secured by irrigating the passages with plain broth. Two-tenths cc. of washings was mixed with 0.1 cc. of bacterial suspension and injected into the flanks of a rabbit. The washings from cases of common cold usually accelerated the reaction to the bacteria more than the washings taken during a normal period. These observations suggested the possibility of a method of increasing the infectiousness of the bacteria in the production of sinusitis. A testicular extract was prepared after the method of Duran-Reynolds from fresh rabbit testicles. The membranes were removed and the testicle ground in sterile Ringer's solution. The supernatent fluid secured after centrifuging at low speed for fifteen minutes was used for injecting. The right sinus of each rabbit was injected with 1 cc. of testicular extract, and followed in a few minutes with vaccine virus plus bacteria. The animals were observed as before. Since only a small quantity of extract was procurable from rabbit testicles, fresh beef testicles were substituted later. The left sinus in each case was injected with saline and used as a control. The rabbits were autopsied, usually after two to four weeks, and the mucous membrane from each maxillary sinus carefully removed and placed in formalin for sectioning. The gross condition of the sinus and membrane was noted. Some were opened and cultured aseptically. A few animals died early and a few were permitted to recover.

DISCUSSION.

A glance at the table will show the greater effectiveness of the testicular extract-virus-bacteria combination over the virus-bacteria mixture in producing sinus infections. All of the eleven animals inoculated in this manner showed definite clinical evidence of sinusitis

Table of Results.

	Virus and Bacteria		Testicular Extract plus Virus plus Bacteria		Bacteria only		Virus only	
	No.	%	No.	%	No.	%	No.	%
Number of cases	34	56.6	11	18.3	11	18.3	4	6.6
Microscopic sections sinus*	25	73.5	8	72.7	8	72.7	2	50.0
Purulent nasal discharge	20	58.8	11	100.0	4	34.6	0	0
Empyema	13	38.2	8	72.7	2	18.2	0	0
Infiltration sinus membrane	16	64.0	8	100.0	2	25.0	2—	
Uninoculated membrane normal	23	92.2	8	100.0	8	100	2	100.0
Bacteria Used:								
Staph. aureus hemolyticus	25	73.5	11	100.0	5	45.4		
Staph. aureus	5	14.7						
plus Bact. lepisepticum					3	27.2		
Strep. hemolyticus	2	5.9			3	27.2		
H. influenza	2	5.9						

*Some cases were not autopsied.

as indicated by the presence of nasal discharge and microscopic evidence of infection was found in all from which the sinuses were examined. There was gross pus in the sinus of every case autopsied, and in all but two the sinus was filled with pus. The left sinus membrane, used as a control, appeared normal in every instance. The regularity of production of infection in this group of animals was quite satisfactory. Chronicity, however, is something which is more difficult to secure, and a certain number of animals will undoubtedly have to be sacrificed to accomplish this end. An occasional one will die of a rapidly fatal infection and others will tend to heal spontaneously. The proper degree of chronicity must be brought about by repeated injections at proper intervals as indicated by the nasal symptoms in each case. Any problem based on the assumption of chronicity must, of course, be thoroughly controlled. All eleven of these animals were injected with a strain of hemolytic staphylococcus aureus, together with testicular extract and vaccine virus, on three successive days. A one day broth culture was used in two cases in place of the usual washings from a blood agar plate without noticeable difference in effect. Two animals were injected with the testicular-extract-virus combination plus supernatent fluid from one day

broth cultures of staphylococcus aureus. There was no evidence of sinus infection, so these animals were not included in the above table. Two other animals used in these experiments were excluded from the table. One died too soon to permit passing judgment as to results and the other developed what appeared to be a spontaneous sinusitis localizing in the noninfected sinus, while the injected side remained free of infection.

The virus-bacteria mixture alone produced gross evidence of sinus infection, as indicated by a dirty nose in 59 per cent of the cases, but only 41.2 per cent of those autopsied showed an empyema of the sinus. A few more, 47 per cent, showed infiltration of the sinus membrane. Some beautiful empyemas were secured by this method, the entire sinus being filled with thick pus, but the results were not as constant or satisfactory as when combined with the testicular extract. There was some evidence of infection in the control sinus in two of these twenty-five cases autopsied. This may be due to crossed infection or spontaneous infection.

The injection of bacteria alone produced some evidence of infection in four out of eleven cases and definite empyema of the sinus in two. It should be pointed out, however, that three of these four cases had a virulent bacterium lepisepticum included in the inoculum. Both cases of empyema were inoculated with this organism. Nasal discharge is the only evidence of infection in the fourth case. The results from injecting alone the same strains of staphylococcus aureus and streptococcus hemolyticus as used in other cases were entirely negative. Bacterium lepisepticum was used in some virus-bacteria mixtures also with evidence of sinus infection in each case. It would appear, therefore, that this organism is particularly virulent for the rabbit.

All animals receiving vaccine virus alone showed only transitory increased moisture about the nose with recovery. There was no sticky, purulent discharge as observed in cases of sinusitis. Symptoms similar to this were observed in some of the animals injected with virus-bacteria mixtures and were classed as noninfected unless there was microscopic or gross evidence of purulent infection.

The question arises as to the nature of the infection induced in these animals. The sinuses of seven rabbits which had been inoculated with hemolytic staphylococcus aureus were opened aseptically and cultured on blood agar. Three of them showed pure cultures of hemolytic staphylococcus aureus from the injected sinus, two a mixture of staphylococcus aureus and bacterium lepisepticum, and two

pure cultures of bacterium lepisepticum. The left, or uninoculated sinus, showed pure cultures of bacterium lepisepticum in two and no growth in the others. The important rôle played by bacterium lepisepticum in all cases of induced sinus infections in rabbits is suggested by these results.

It should be pointed out that the quantity of vaccine virus used in all these cases was quite small. Increasing the quantity to an optimum may give more consistent or more desirable results for experimental purposes.

SUMMARY.

1. The use of the rabbit as an experimental animal in the study of sinusitis has been investigated.

2. Purulent sinusitis could be produced with considerable regularity by the injection of a vaccine virus-bacteria-testicular extract combination into the maxillary sinus of the rabbit.

3. Results were secured principally by the use of strains of hemolytic staphylococcus aureus or staphylococcus aureus plus bacterium lepisepticum.

4. This method of procedure simulates the natural mode of infection which frequently results in sinusitis in the human.

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XCIX.

ACUTE SUPPURATIVE OTITIS MEDIA IN MEASLES: A REPORT OF 427 PATIENTS.

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The statement will be accepted without challenge, it is believed, that among the specific infectious diseases scarlet fever and measles stand out far above all the others in importance in the production of acute suppurative otitis media, and that of these two diseases the relative importance, both as to frequency and severity, is scarlet fever first and measles second.

Scarlet fever is considered a major contagion and measles a minor one, and from the standpoint of public health problems the method of handling scarlet fever differs from that of managing measles. An attempt is made to hospitalize practically all patients with scarlet fever, and the quarantine period usually is a minimum of thirty days. On the other hand, regarding patients with measles, no attempt is made to hospitalize other than selected patients. These latter fall chiefly into the following groups:

(a) Patients who are inmates of a hospital when they develop the disease.

(b) Patients who are charges of various kinds of homes and nurseries.

(c) Patients who are members of various kinds of boarding schools.

(d) Patients who develop some complication, such as meningitis, encephalitis, bronchopneumonia, mastoiditis or appendicitis, requiring hospital facilities.

(e) Patients who are too poor to afford a private physician; and a few patients who for some reason not above mentioned, desire and secure hospital treatment.

The quarantine period for measles is usually a minimum of about two weeks.

During the decade of 1922-1931, inclusive, there were 37,873 cases of scarlet fever and 92,745 cases of measles reported in Philadelphia. During the same period of time there were treated at the Philadelphia Hospital for Contagious Diseases, the city's only hospital for the care of the so-called contagious diseases, 23,804 patients with scarlet fever and 1,954 patients with measles. Therefore, 62.6 per cent of the city's scarlet fever patients and 2.1 per cent of its measles patients were hospitalized.

During the aforementioned decade (1922-1931) there were 2,168 scarlet fever patients in the Philadelphia Hospital for Contagious Diseases who suffered from acute suppurative otitis media, while during the same period there were only 427 measles patients who had this affection.

These data are set forth here in the beginning so that one may determine to what degree hospital statistics are representative of measles statistics in general, and to enable one properly to study the following figures, especially those that refer to the Philadelphia Hospital for Contagious Diseases. It seems probable that these figures are not representative of measles as found in general practice, and that they will have to be considered for just what they are, namely, hospital statistics.

The incidence of acute suppurative otitis media in hospitalized measles patients varies widely. (See Table I.) Among the aforementioned 1,954 measles patients there were 427 (22 per cent) who suffered from acute suppurative otitis media while in the hospital.

Reported in Table I are comparative figures from Cody,[2] Dixon,[3] and Gardiner.[1]

TABLE I.

Author	Number of Measles patients	Number of Ac. Sup. Otitis Media	Per cent. Otitis Media in Measles
Cody	1769	321	18.2
Dixon	220	103	47.0
Gardiner	1331	181	13.5

These 427 patients with acute suppurative otitis media at the Philadelphia Hospital had 651 affected ears. Practically half of them, therefore, had both ears involved. In 321 of the ears the tympanic membrane was incised before it ruptured, 36 after it had ruptured spontaneously; and in 294 the tympanic membrane ruptured spontaneously and was not incised. Among those in which the tympanic membrane was incised before it ruptured there were 13 (4.4 per

cent) mastoidectomies; among those in which the drum ruptured and was not incised there were 33 (11.3 per cent) mastoidectomies; and among the small group of 36 ears in which the tympanic membrane was incised after it had ruptured there was one mastoidectomy. (See Table II.)

TABLE II.

Ears		Patients receiving mastoidectomy Unilateral	Bilateral
Incised	321	12	1
Ruptured and Incised	36	1	0
Ruptured	294	21	6
Total number of ears			651
Number of patients receiving mastoidectomy			41
Number mastoidectomies performed			48

If one be permitted to draw conclusions from such figures as, let us say, the two larger groups, he would have to declare that early incision of the tympanic membrane in acute suppurative otitis media in measles has a distinct tendency to prevent the development of surgical mastoiditis.

The number of patients undergoing mastoidectomy was 41, or 9.6 per cent of the 427, with acute suppurative otitis media.

Table III gives the comparative figures of Cody,[2] Dixon,[3] and Gardiner.[1]

Because measles patients are quarantined only about two weeks and because those patients who have been operated on for mastoiditis are discharged before the wound has healed, the hospital records do not contain data on the length of time required for healing.

TABLE III.

Author	Number measles patients with Ac. Sup. Otitis Media	Number with Mastoiditis	Percentage developing mastoiditis
Cody	321	29	9
Dixon	103	34	33
Gardiner	181	7	4

The organisms most frequently isolated from mastoiditis complicating measles are streptococcus[1] (hemolyticus and pyogens) and staphylococcus.[5] Pneumococcus more rarely is found.[3] These organisms are usually found in pure culture.

The incidence of otitis in measles according to age is highest in the young. Of the 427 patients here considered 332 (77.7 per cent) were under 6 years of age; 64 (15 per cent) were between the ages

of 6 to 10; 9 (2.1 per cent) were between the ages of 11 and 15; 6 (1.4 per cent) were between the ages of 16 and 20; and 16 (3.7 per cent) were more than 20 years. (See Table IV.)

Of Gardiner's 181 patients with otitis 96 were under two years of age. The mean age of Cody's 1,769 measles patients was 4.81 years. Dixon's series of 220 measles patients is unusual. It was composed of young male adults averaging 19 years of age. One hundred and three (47 per cent) of them developed otitis media, and 34 (15 per cent) developed mastoiditis. The incidence of this complication in the 103 patients with otitis media was 33 per cent. (See Table III.) This incidence as regards both otitis media and mastoiditis is the highest of any series encountered.

TABLE IV.

Age	Number of Patients	Percentage
0–5	332	77.7
6–10	64	15.0
11–15	9	2.1
16–20	6	1.4
21–	16	3.7

Nearly all of the patients in this series had tonsils and adenoids intact. When one considers the age incidence of the group the probable reason is apparent: 77.7 per cent of them were under six years of age. Among the 427 patients tonsils and adenoids were present in 418 and absent in nine. Of those who developed mastoiditis forty had tonsils and adenoids and one had none. (See Table V.)

TABLE V.

	Patients with tonsils and adenoids	Patients who developed mastoiditis
Present	418	40
Absent	9	1

CONCLUSIONS.

If one be permitted to draw conclusions from this material, it may be said that:

1. The incidence of acute suppurative otitis media in hospitalized measles patients is approximately 22 per cent.

2. Approximately half of the number of patients affected with otitis in measles have a bilateral affection.

3. Early myringotomy in acute suppurative otitis media in measles has a distinct tendency to prevent the development of surgical mastoiditis.

4. Approximately 10 per cent of the patients who suffer from acute suppurative otitis media in measles develop surgical mastoiditis.

5. Seventy-seven and seven-tenths per cent of the patients with otitis in measles are under six years of age.

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C.

STREPTOCOCCUS HEMOLYTICUS BACTEREMIA WITH SPECIAL REFERENCE TO OTOLARYNGOLOGIC CONDITIONS.*

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In the five and one-half years with which this study is concerned there have been 168 cases of streptococcus hemolyticus bacteremia observed in the various medical and surgical departments of the Mount Sinai Hospital. This constitutes about one-fifth of all proved bacteremias during the stated period. Of these cases, 70, or 42 per cent of the total number, were of either otitic or pharyngeal origin.

Because of the comparatively high incidence of streptococcus hemolyticus bacteremias and the clinical importance of such blood stream infections, we felt that a comprehensive study of these cases would be both interesting and valuable. In this study we tried to classify the various conditions in which streptococcus hemolyticus bacteremia occurred and to determine the significance of the bacteriologic findings in each group of cases. The route of invasion into the blood stream was used for the grouping of the cases. Each group was further studied in relation to age, seasonal incidence, prognosis and the clinical picture observed. In analyzing the blood cultures we were especially interested in the diagnostic and prognostic significance of the number of organisms cultivated from the blood stream.

METHODS.

The bacteriologic findings in the cases which formed the basis of this report were studied in our laboratories by means of uniform and standard methods. The blood culture technic employed in these

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cases, which has been described in complete detail elsewhere,[1] was as follows:

Twenty-one cubic centimeters of the patient's blood were withdrawn from an anterior cubital vein and mixed with the media at the bedside. Fifteen cubic centimeters were divided equally among three 100 cc. Erlenmeyer flasks, the first containing veal infusion broth, pH 7.2; the second, 2 per cent glucose veal infusion broth, pH 7.2; and the third, 10 per cent tomato extract veal infusion broth, pH 7.2. The remaining 6 cc. of blood were equally divided to form three pour plates, 2 cc. being mixed with a tube of 12 cc. of liver hormone veal infusion agar, pH 7.2, 2 cc. with a tube of 12 cc. of 2 per cent glucose veal infusion agar, pH 7.0—7.4, and the same amount with a tube of 12 cc. of plain veal infusion agar, pH 7.2, respectively.

TABLE I.

BLOOD CULTURE DIVISION.

21 CC. OF BLOOD.

15 cc. for Fluid Media			6 cc. for Solid Media		
5 cc. Veal Infusion Broth 90 cc. pH 7.2	5 cc. 2% Glucose Veal Infusion Broth 90 cc. pH 7.2	5 cc. 10% Tomato Extract Veal Infusion Broth 90 cc. pH 7.2	2 cc. Liver Hormone Veal Infusion Agar 12 cc. pH 7.4	2 cc. 2% Glucose Veal Infusion Agar 12 cc. pH 7.0-7.4	2 cc. Veal Infusion Agar 12 cc. pH 7.2

Each of the 100 cc. Erlenmeyer flasks contained 90 cc. of fluid medium. In this manner a high degree of partial anaerobiosis was obtained as a result of the small surface exposed to oxygen as compared to the large volume of the medium.

Daily transplants were made for three days from each of the flasks onto slants of ascitic glucose fluid agar and also into tubes containing glucose veal infusion broth. In addition, smears stained by the Gram method were examined daily from each flask containing fluid media.

The streptococcus hemolyticus (streptococcus Beta-Brown) was identified by the appearance of the growth of transplants on solid media; by the hemolytic effect on the red blood cells on blood agar plates; by the retention of the Gram stain; and by the chain formation of the cocci in fluid media. The criteria for identification sug-

gested by Brown were followed. In addition, all isolated gram positive cocci were differentiated from pneumococci by the test of bile solubility and serological methods.

MATERIAL.

In order to classify our material, the term portal of entry was used to indicate the actual site from which the streptococcus hemolyticus gained entry into the blood stream. This site need not coincide with the original focus of infection. Thus, an infection may spread by contiguity for a considerable distance from its original site and finally invade the blood stream via a "portal" somewhat removed from the primary infection. Distant foci developing during the course of the bacteremia were termed "secondary" or metastatic foci. These cases were grouped, whenever possible, according to the portal of entry. Thus, the following classification, arranged in order of mortality rates, was formulated:

TABLE II.

Group	Cases	Mortality
I. Secondary Erysipelas	5	20%
II. Upper Respiratory Infections	23	34%
III. Peripheral Infections	22	36%
IV. Lateral Sinus Thrombosis	43	37%
V. Primary Erysipelas	2	50%
VI. Gynecological Infections	10	60%
VII. Osseous and Articular Infections	8	62%
VIII. Associated Non-bacterial Diseases	23	74%
IX. Surgical Postoperative Infections	20	85%
X. Acute Otitis Media with Meningitis	4	100%
XI. Pulmonary Infections	8	100%
Total	168	54%

We shall present in this paper the groups of otolaryngologic interest.

UPPER RESPIRATORY INFECTIONS.

By the term upper respiratory infections is meant infections of the sinuses, nasopharynx, pharynx or larynx. There were twenty-three patients in this group, of whom eight died and fifteen recovered. This low mortality of 34 per cent attracted attention because of the contrast to the much higher death rates of streptococcus hemolyticus infections originating from other foci. In twenty the infection was diagnosed as a pharyngitis or nasopharyngitis without pulmonary involvement. The remaining three cases included two cases of tonsillitis, one with a peritonsillar abscess, and one case of post-tonsillec-

tomy infection. The above described conditions were the only primary foci which could be discovered clinically. There was no bacteremia originating from an uncomplicated sinus infection or the larynx in this series of cases.

Fifteen patients were between the ages of 1 and 5, six between 5 and 20 years, and the remaining two were 40 and 72 years old, respectively. There was, thus, an unquestionable predilection for the early years of life. Only three cases occurred during the summer and two in the fall (November). The remaining eighteen cases occurred during the winter and early spring (March and April). This group, therefore, also showed a definite seasonal incidence. In connection with the opinion that tonsillectomized patients are more prone to develop a general infection in the presence of a pharyngitis, it is of interest to mention that of the twenty cases, only eight had a previous tonsillectomy.

We were strongly impressed by the fact that ten of the twenty-three patients in this group developed osteomyelitis of the long bones, and of these, only one died. The nine recovered patients were under 6 years of age, and the patient who died was 11. In each instance only one bone was involved and the osteomyelitis was either the first or only complicating condition. In these cases the osteomyelitis was undoubtedly the source for the persistent bacteremia.

In most of the fatal cases the blood culture showed a considerable number of streptococci present in the blood stream. In the fifteen recovered cases, eleven blood cultures were positive in fluid media only and four in all media. Of the eleven just mentioned, five showed growth in only one of the fluid media. It is interesting that in the blood cultures of the nine patients who developed osteomyelitis and recovered, organisms grew in fluid media only in six instances.

In this group of twenty-three cases there was a history of an upper respiratory infection in every instance preceding the discovery of the bacteremia. In most of the cases a local inflammatory process was still evident when a septic clinical picture appeared. Thus, this group of cases presents convincing evidence that an upper respiratory infection may be the source of a streptococcus hemolyticus bacteremia. The persistence of the bacteremia in nineteen of these cases, however, might be attributed to metastatic foci or contiguous infections. In only four cases the infection remained entirely localized (one case of tonsillitis with peritonsillar abscess and three cases of pharyngitis in young children).

In twenty of the cases a pharyngitis or nasopharyngitis was apparently the portal of entry for the organism. Whether the bacteria gained entry into the blood stream by way of a local phlebitis or by way of the regional lymphatics cannot be ascertained as yet. This question requires further pathologic investigation.

Thus, we are dealing in this group with a type of streptococcus hemolyticus bacteremia originating in the upper respiratory tract and occurring for the most part in children during the winter and spring months. Apparently, these blood stream infections have a comparatively favorable prognosis and show a definite metastatic predilection for long bones.

LATERAL SINUS THROMBOSIS.

Data as to blood cultures in this group of cases are presented for a longer period of seven years. During this time, operations for lateral sinus thrombosis were performed on sixty-three patients. The results of preoperative blood cultures were as follows:

In two cases no blood cultures were taken; in two the blood cultures were reported sterile; in one, bacillus pyocyaneous was isolated; in another, bacillus proteus; and in fifty-seven cases streptococcus hemolyticus was found in the blood stream in variable numbers. Thus, the preoperative blood cultures were positive in 96.7 per cent of the patients. The large amount of blood taken for cultivation, the employment of a variety of solid and fluid media, and repeated blood cultures were all important factors in obtaining this high incidence of positive blood cultures. In view of the practical importance of a positive blood culture in the diagnosis and operative indication of lateral sinus thrombosis, these figures are summarized in Table III.

TABLE III.

Distribution of Positive Blood Cultures in Cases of Lateral Sinus Thrombosis According to the Media in which Growth was Obtained.

Preoperative Blood Cultures				Postoperative Blood Cultures			
Fluid Media Only			All Media	Fluid Media Only			All Media
1 Medium[1]	2 Media[2]	3 Media[3]		1 Medium[1]	2 Media[2]	3 Media[3]	
14	4	15	33	2	3	10	17
Total—66				Total—32			

[1] Usually tomato or glucose broth.
[2] Usually tomato and glucose broth.
[3] Tomato, glucose and plain broth.

As is seen from Table III, bacterial growth occurred in fluid media alone in 50 per cent of the preoperative blood cultures. Of these positive cultures, 21.2 per cent showed organisms in only one of

the fluid media. Of the postoperative blood cultures, bacterial growth was restricted to fluid media in 44 per cent of the cultures and to one fluid medium in 13 per cent of the cultures.

Further analysis of the group concerns only forty-three of the above sixty-six cases which occurred during the five and a half year period originally considered in this presentation. Of these, twenty-seven recovered and sixteen died, a mortality rate of 37 per cent. Thirty-three of the patients were less than fifteen years of age and none were over fifty. Thus, there was a predilection for the early years of life. There was no predominant seasonal incidence. The main metastatic foci in this group of cases occurred in the kidneys, lungs, bones and joints. Intracranial involvement was the most common fatal complication.

Libman's original blood culture work[2] on lateral sinus thrombosis is one of the great contributions to otology. He demonstrated that a frank bacteremia is an important and constant feature in the clinical picture of lateral sinus thrombosis and that the invading organism is usually streptococcus hemolyticus.

Our studies of a larger group of these cases have once more corroborated his findings with a higher percentage of positive results. Thus, of sixty-three patients operated on for lateral sinus thrombosis, a preoperative blood culture was taken in sixty-one cases and found positive in fifty-nine instances (96.7 per cent). Of these fifty-nine proven bacteremias, streptococcus hemolyticus was obtained from fifty-seven cases (96.6 per cent).

It is well known that the number of bacteria in the blood stream during the course of a general infection may vary greatly at different times the day. The report of a sterile culture on one occasion and numerous organisms per cubic centimeter of blood from the same patient on the following day is not an infrequent occurrence in the bacteriology laboratory. The need, therefore, for repeated blood culture studies in suspected cases of lateral sinus thrombosis, where the diagnosis is so dependent on these cultures, cannot be overemphasized.

Since these facts concerning lateral sinus thrombosis have been fully appreciated, blood culture studies have been especially helpful in solving septicemias with vague otitic histories and inconclusive physical findings. The discovery of streptococcus hemolyticus in such cases has led to the diagnosis of lateral sinus thrombosis with consistent accuracy, after all other sources of infection have been logically excluded. Conspicuously illustrative of such instances is the type of sinus thrombosis known as primary jugular bulb thrombosis.[3]

These patients when examined usually presented a healed or healing middle ear infection and no evidence of mastoiditis. The history of a recent otitic infection and the presence of a septicemia suggested the possibility of a primary jugular bulb thrombosis, but the finding of a hemolytic streptococcus blood stream infection established the diagnosis when no other focus of infection could be ascertained clinically.

We believe that the presence of even a very small number of hemolytic streptococci in the blood stream at any time is indicative of a frank bacteremia. In other words, in a suspected case of lateral sinus thrombosis, growth of streptococcus hemolyticus in fluid media only, even on one occasion in a single flask, is suggestive of a sinus wall infection, all other clinical possibilities having been reasonably considered and excluded. The same view may be entertained for pneumococcus type III, but for no other organism. Before any other bacterium is regarded as the causative agent, it should be isolated from both solid and fluid media on one occasion, or from fluid media on two occasions. As was mentioned above, bacterial growth occurred in fluid media alone in 50 per cent of the preoperative blood cultures, and of these positive blood cultures, 21.2 per cent showed organisms in only one of the three fluid media. It is apparent, therefore, that if the amount of blood taken for cultiyation had been reduced or some of the media eliminated, the incidence of positive blood cultures would have been considerably diminished. If solid media alone had been used, the incidence of positive cultures would have decreased by one-half. It is also evident that in at least 50 per cent of the cases the number of organisms present in the blood stream preoperatively was very small. For this reason it is at times necessary to take repeated blood cultures.

Repeated sterile cultures, we also believe, represent information which should be seriously considered in the evaluation of cases under observation. An otologist who has reliable bacteriologic studies at his disposal should be disinclined to operate on a suspected case of sinus thrombosis with repeated sterile blood cultures, unless undeniable clinical findings are encountered. This policy has borne satisfactory results, for such cases after a period of observation have evidenced manifestations which indicated the presence of a pneumonia, pharyngeal abscess, pyelonephritis, primary meningitis, or some other illness which accounted for the clinical course.

ACUTE OTITIS MEDIA WITH MENINGITIS.

There were four cases of acute otitis media with meningitis which showed numerous streptococci in the blood stream. They all

resulted fatally. Inasmuch as middle ear infections alone do not produce any bacteremia, the meningitis had to be considered as the source of the blood invasion.

All the cases occurred in the winter and spring. In three cases there were no secondary foci, most likely because of the fulminating course.

DISCUSSION.

In presenting the following discussion, we have taken into consideration all the groups. In this way the otolaryngologic groups can be evaluated and appreciated in relationship to the other bacteremias.

These cases of streptococcus hemolyticus bacteremia showed a close correlation between bacteriologic findings and clinical significance. We have observed that the occurrence of a positive blood culture with the technic employed indicated a general infection (septicemia) clinically. In this series no positive blood cultures were encountered in cases where such a general infection was absent.

It should be noted that certain features in our blood culture technic were apparently responsible for the high percentage of positive blood cultures obtained. We refer particularly to the large quantity of blood drawn, the variety and enrichment of the media employed, the careful adjustment of pH of the media, the daily subcultures, spreads and prolonged observation. The sensitivity of our routine media has been checked at various times by simultaneous comparison with a number of other recommended media, including those of Cecil, Nichols and Stainsby,[4] the Kendall media[5] and various anaerobic methods,[6] and none of these showed a higher incidence of positive blood cultures.

The efficacy of our blood culture methods has been demonstrated by our experiences with diseases proven to be associated with bacteremia. Thus, in lateral sinus thrombosis, a condition in which the blood stream is invaded at some time during the course of the disease, positive blood cultures were obtained in 96.7 per cent of the cases over a seven year period. Also during a similar period with which this study is concerned, in 95.5 per cent of the cases of subacute bacterial endocarditis which came to autopsy, nonhemolytic streptococci had been cultivated from the blood stream by the same technic.[7]

We have been especially interested, while studying these blood cultures, in the clinical significance of the growth of streptococcus

hemolyticus in fluid media alone as distinguished from growth in both fluid and solid media. We have been able to establish certain diagnostic and prognostic criteria from our observations.

Growth in both fluid and solid media was interpreted to indicate the presence of large numbers of organisms in the blood stream, whereas growth restricted to fluid media indicated a paucity of organisms. This becomes clear when we consider that inoculation of a single bacterium into a fluid medium yields a high concentration of bacteria in twenty-four to forty-eight hours. On the other hand, similar inoculations into a solid medium will yield only a single colony. Furthermore, the initially inoculated bacterium is much less likely to survive on a solid medium than in a fluid medium.

The most striking observation which this study presents was that every positive blood culture of streptococcus hemolyticus was associated with clinical indications of blood invasion. In other words, every case was a "frank" bacteremia and clinically significant, while what is known as a "transitory" and clinically insignificant bacteremia with no evident portal of entry was not encountered. This also applied to cases in which growth was limited to fluid media, even to one fluid medium. Thus, in the cases of peripheral infection, upper respiratory infection and lateral sinus thrombosis, conditions in which a positive blood culture was very important for diagnosis, 48 per cent of the blood cultures were positive in fluid media only. Furthermore, in 36 per cent of these positive blood cultures growth occurred in one fluid medium. In every one of these cases the blood stream was being fed by a demonstrable septic focus.

Growth of streptococcus nonhemolyticus (Alpha and Gamma-Brown) confined to fluid media does not have the same clinical implication as indicated above for streptococcus hemolyticus. Such a bacteremia might belong to the transient variety and be clinically unimportant. Such observations have been made by Libman,[7] Lichtman and Gross,[8] Epstein and Kugel,[9] Burn,[10] and confirmed experimentally by Desoubry and Porcher.[11]

It is interesting from a prognostic consideration that the groups which had the larger percentage of positive blood cultures in fluid media only, showed the lowest mortality rates (peripheral infections, upper respiratory infections, lateral sinus thrombosis). Even in the groups with high death rates the recovered cases were usually those whose blood cultures showed growth restricted to fluid media (gynecologic infections, associated nonbacterial diseases). The high mortality associated with the cases of acute otitis media with meningitis

and pulmonary infections (100 per cent in our series) is well known. Of special interest, because of its clinical importance and comparatively frequent occurrence, is the group of surgical postoperative infections which presented the third highest mortality rate of 85 per cent.

TABLE IV.

Relationship of Quantitative Number of Organisms to Mortality Rates (Prognosis).

Group	Total Cases	Total Mortality	No. of fatal cases	Fatal Cases positive in all Media	No. of recovered cases	Recovered cases positive in Fld. Media
Upper Respiratory Infections	23	34%	8	5	15	11
Peripheral Infections	22	36%	5	5	17	10
Lateral Sinus Thrombosis	43	37%	16	9	27	15
Gynecological Infections	10	60%	6	6	4	4
Associated Non-Bacterial Diseases	23	74%	17	17	6	4
Pulmonary Infections	8	100%	8	8		

SUMMARY.

In this study 168 cases of streptococcus hemolyticus bacteremias were described and classified according to the portal of entry of the organism.

Of the 168 patients, 91 died—a mortality rate of 54 per cent. A mortality rate ranging from 60 to 100 per cent was encountered in the following groups: gynecologic infections, articular and osseous infections, miscellaneous nonbacterial conditions associated with streptococcus hemolyticus, surgical postoperative infections, pulmonary infections and acute otitis media with meningitis. In contrast to these, the mortality rate of the cases of secondary erysipelas was 20 per cent, upper respiratory infections 34 per cent, peripheral infections 36 per cent, lateral sinus thrombosis 37 per cent, and primary erysipelas 50 per cent.

Ninety-one per cent of the cases in which the primary foci were located in the respiratory tract (pulmonary infections, upper respiratory infections, acute otitis media with meningitis) occurred in the winter and spring months. The remaining conditions manifested no particular seasonal influence.

Upper respiratory infections, lateral sinus thrombosis and osseous and articular infections showed a tendency to occur during the early years of life. The other conditions evidenced no predilection for a

special age. In the group of peripheral infections the mortality during the middle years of life was considerably higher than during the earlier years of life (75 per cent as compared to 25 per cent).

The enrichments of the blood culture media and the blood culture methods employed were largely responsible for the high incidence of these positive streptococcus hemolyticus blood cultures, especially when limited to fluid media.

The quantitative estimation of the number of hemolytic streptococci in the blood stream (i. e., growth of the bacteria in both solid and fluid media or in fluid media alone) had both diagnostic and prognostic significance.

The conspicuous groups illustrating the diagnostic value of these blood cultures were the cases of lateral sinus thrombosis, upper respiratory infections and peripheral infections. In the cases of lateral sinus thrombosis, 50 per cent of the preoperative blood cultures showed growth in fluid media only, and growth appeared in but one fluid medium in 21 per cent of these cultures. In the cases of upper respiratory infections and peripheral infections, bacterial growth was restricted to fluid media in 61 per cent and 45 per cent, respectively, of all cultures.

The prognostic import of these blood culture results is demonstrated by the fact that the groups with relatively low mortality presented a high percentage of positive blood cultures in fluid media only, while the groups with relatively high mortality had a high percentage of positive blood cultures in both solid and fluid media. It is significant that all the gynecologic and associated nonbacterial cases showing growth in fluid media only recovered.

Of special interest among the cases of upper respiratory infection was the group which developed long bone metastases. The infection in these cases (ten) manifested a predilection for young children, a tendency towards complete recovery (90 per cent), and a small number of organisms in the blood stream in a majority of the cases (fluid media only 60 per cent).

The data embodied in this paper disclose that in contrast to nonhemolytic streptococci (Alpha and Gamma), the finding of streptococcus hemolyticus (Beta) in the blood stream, even in extremely small numbers, was of important clinical significance for diagnosis, prognosis, and indication for surgical interference.

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CI.

ACTINOMYCOSIS OF THE SPHENOID WITH ACTINOMYCOTIC MENINGITIS AND BRAIN ABSCESS.*

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In extensive studies of the bacteriology of accessory nasal sinus suppuration by Turner,[1] no mention is made of actinomyces as a primary pathogenic organism. However, secondary involvement of the accessory nasal sinuses, particularly the antra of Highmore, may result from extension of fungus infections in dental caries of the upper jaw. Likewise, actinomycotic infection of the nasopharynx may spread to the paranasal sinuses, as in two instances cited by New and Figi.[2] Another possible source of secondary actinomycotic infection of the paranasal sinuses is that resulting from primary actinomycotic mastoiditis. Beysiegel[3] collected fifteen instances of actinomycotic mastoiditis, and in the majority of these he believed that the infection originated in the external auditory canal and subsequently involved the mastoid and paranasal sinuses. In several instances, however, he assumed the existence of a primary actinomycotic nasopharyngitis with secondary extension to the mastoid by way of the eustachian tube and middle ear.

The only reported instance of primary actinomycotic infection of the sphenoid sinus is that of Stevenson and Adair Dighton.[4] They observed a case of meningitis following enucleation for pneumococcic panophthalmitis resulting from traumatic ulceration of the cornea. The authors assumed that the meningitis was secondary to the panophthalmitis. At autopsy the meningitis, however, was found to be the result of a primary actinomycotic osteomyelitis of the sphenoid sinus with intracranial extension.

In the case here reported, the diagnosis of sphenoiditis was entertained during life and the patient operated upon intranasally, although the bacteriology was not determined until after death.

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CASE REPORT.—A 25-year-old Hungarian woman was admitted to the laryngological service of the Mount Sinai Hospital on September 20, 1934. She had been in good health until three weeks prior to admission. At that time she developed an upper respiratory infection and several days later complained of severe right frontal headache radiating behind the right ear to the occiput and to the nape of the neck. Nasal obstruction and a moderately profuse postnasal discharge developed. There was no history of chills and the daily temperature was never elevated above 99° F. Because of persistent headache and vomiting she was admitted to the hospital.

A physical examination revealed no significant findings, except for the local nasal condition. Intranasal examination revealed a deviated septum with intense congestion of the nasal mucosa, more marked on the right side. There was thick mucopus in the right olfactory sulcus and middle meatus. On posterior rhinoscopy, purulent exudate was seen in the posterior choanæ. No evidences of meningeal irritation were observed. A diagnosis of acute pansinusitis with sphenoid involvement was made and the patient was treated with nasal sprays of cocaine and adrenalin followed by steam inhalations.

Course.—Severe occipital pain persisted despite increased drainage from the sinuses. A blood count showed a hemoglobin of 75 per cent Sahli, 12,000 leucocytes with 76 per cent polymorphonuclear neutrophiles and 24 per cent lymphocytes. Fundus examination revealed reddening of the nerve head on the right with indistinct margins, but no real edema. The retinal vessels were slightly full. An x-ray examination of the paranasal sinuses showed a small right sphenoid sinus which was definitely cloudy. The left sphenoid sinus was considerably larger and also hazy, as was the antrum on the same side (Fig. 1).

Irrigation of both sphenoids through the natural orifices yielded a moderate amount of mucopus from the right side and a clear return from the left. Low grade fever, pain in the nape of the neck and tenderness over the right occiput persisted. The tip of the spleen was now palpable, and the liver could be felt two fingers below the costal margin. Agglutination tests for typhoid, paratyphoid and melitensis, and repeated blood cultures were negative.

Four days after admission, slight spasticity of the neck was detected. This could be easily overcome, although the patient winced when the head was flexed on the chest. No Kernig or Brudzinski was present. Lumbar puncture was performed and a small quantity of clear, transparent cerebrospinal fluid was withdrawn. The manometric

Fig. 1. Roentgenogram of nasal sinuses. Right sphenoid considerably smaller and distinctly more cloudy than left sphenoid.

pressure was 114 mm. of water, the cell count showed one lymphocyte per cubic mm. Culture of the spinal fluid was sterile. An area of fullness and exquisite tenderness was now noted over the right occipital region, just behind the sternomastoid muscle. X-ray examination of the cervical spine showed no abnormality.

A right spheno-ethmoidectomy with intranasal antrotomy was performed with the Yankauer technic, under local anesthesia. The right sphenoid sinus was found to be partially filled with a viscid, glairy secretion but did not contain free pus. The mucosa of the floor of the sphenoid sinus was thickened, irregular and polypoid. The mucosa was not stripped from the bone.

The intranasal operative procedure had no effect upon the course of the illness. Headache, occipital pain, and nuchal tenderness with intermittent fever of 103° F. continued. Cervical tenderness now shifted to the region of the atlas. Re-examination of the cervical spine roentgenographically again failed to reveal any abnormality. The diagnosis of osteomyelitis of the cervical vertebrae secondary to infection of the basi-sphenoid was suggested and the spine immobilized with a plaster collar. This procedure caused no subsidence of pain.

On October 4th, nine days after ethmoidectomy, the patient developed severe pain over the left frontal region and occiput, radiating to the neck. Bony tenderness was now more manifest on the left side of the neck, although still present on the right. The posterior cervical chain of lymph glands became palpable and tender. Neurologic examination at this time still showed no signs of central or peripheral nervous system involvement. The course of the patient's illness continued unabated. Shock tenderness developed over the dorsal spine and left costovertebral angle. Examination of the urine was negative, the leucocyte count was 21,300 with 90 per cent polymorphonuclear neutrophiles. A striking feature in the relative disproportion between the rate of the pulse and the height of the fever was now noted. With a temperature of 102° F., the pulse varied between 80 and 88 per minute. The plaster cast had proved of no benefit and was removed. Definite limitation of flexion of the head with spasm of the cervical muscles was present. A spinal tap was performed and turbid fluid obtained under an initial pressure of 260 mm. of water. This promptly rose to 520 mm. on jugular compression. Cell count of the spinal fluid showed 380 cells per cubic mm. with 85 per cent polymorphonuclear neutrophiles and 15 per cent lymphocytes. A centrifuged specimen showed no organisms on smear and the culture was sterile. Cisternal puncture was performed and yielded a ground glass fluid containing 1900 cells with 88 per cent polymorphonuclear neutrophiles. Globulin was present. On standing, the fluid formed a pellicle, smear of which showed no acid fast bacilli. The Wassermann test and colloidal gold were negative; the fluid contained 25 mgm. of sugar, 595 mgm. of chlorides, total of proteins 90 mgm. per 100 cc.

The patient became apathetic and on the 20th of October developed a bilateral papilledema, right facial weakness, absent right abdominal reflexes, a positive right Babinski, patellar and ankle clonus. bilateral Kernig and rigidity of the neck. The visual fields could not be tested, because of the patient's inability to co-operate. In addition, there was tenderness on percussion over the left temporal region of

the skull. The diagnosis of left temporal lobe abscess was made. On the following day the patient became more drowsy, totally aphasic, with increased blurring of the discs. A craniotomy was performed and a button of bone was removed from the left temporal region. The dura was normal in appearance, and the brain could be seen shining through it, under increased pressure. On introducing the needle downward and forward, nonresisting tissue was entered and aspiration yield disorganized brain with pus droplets. The patient died forty-eight hours after operation.

A complete postmortem examination was performed by Dr. Silverman and Dr. Tarachow.

General Examination: There were multiple lung abscesses. These consisted of small spherical nodules located subpleurally and on section showed necrotic centers. The abscesses were sharply demarcated from the surrounding lung parenchyma. Aside from passive congestion of spleen, liver and kidney, the remaining viscera showed no significant alteration.

Microscopic examination of nodules in the lung showed them to be composed of broken down pulmonary parenchyma which was partly replaced by red blood cells and masses of polymorphonuclear leucocytes. The abscesses were surrounded by congested, collapsed alveoli which contained many histiocytes. Bacterial stains of these sections showed gram positive cocci in chains. Actinomycotic colonies were not found.

Brain (Examined by Dr. J. H. Globus):

Gross Anatomy.—The dura was under moderate tension. There was a massive green purulent exudate at the base of the brain. The ventral surface of the brain stem was covered by the fibrinopurulent, greenish exudate extending caudally as far as the level of the decussation of the pyramids and rostrally to the level of the temporal poles. It coated the restiform bodies, brachia pontis and lay thick over pons, cerebral peduncles, mamillary bodies, optic tracts, chiasm and optic nerves. The right lateral fissure was free; the right temporal pole could be separated readily from the overlying frontal lobe. On the left, however, the exudate obliterated the lateral fissure, extended beyond the temporal pole, and bound the temporal and frontal lobes together.

Section through the left temporal lobe revealed an abscess cavity at the anterior pole (Fig. 2). It measured approximately 2 x 2 x 2 cm. and possessed an irregular but well defined wall. The tract result-

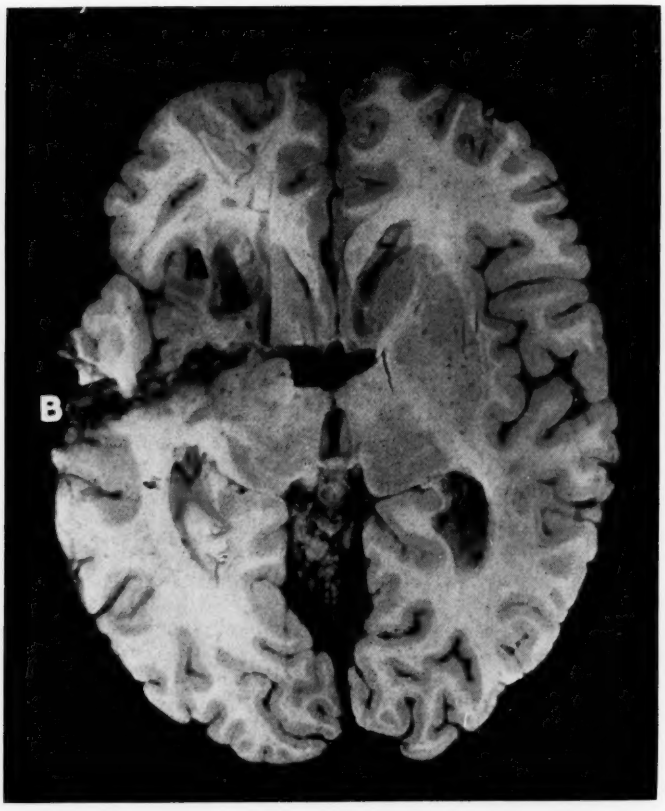

Fig. 2. Photograph of horizontal section of brain. (A) Abscess at anterior tip of the left temporal lobe. (B) Tract due to exploratory puncture caudal to cavity.

ing from the operative exploration of the temporal lobe abscess did not enter but was caudal to it. The cavity contained thick, greenish, flaky exudate. At its medio-anterior border the wall became less than 1 mm. thick and broke into the lateral fissure about 1 cm. rostral to the uncus. This abscess did not communicate with the inferior horn. Section through the left frontal lobe revealed another abscess lying in the midportion of the frontal lobe immediately above the site of the temporal abscess. This cavity measured approximately 1.5 x 1.5 x 1.5 cm. and presented an irregular well defined wall and contents similar to that found in the temporal abscess. The roof of this cavity lay 0.5 cm. lateral to the antero-inferior tip of the anterior horn and

did not communicate with the ventricular system. The abscess occupied the site of the anterior half of the anterior limb of the internal capsule, part of the caudate nucleus and part of the anterior portion of the lentiform nucleus reaching laterally to the gray matter of the cortex. Its wall was thinnest (approx. 1 mm.) at its inferior aspect, where it lay in contact with the lateral fissure immediately above the site of the perforation of the temporal abscess.

It would seem that both the temporal and the frontal lobe abscesses had a common point of origin. A single vascular trunk through its branching acted as the vehicle of the infective material.

Microscopic Anatomy.—Sections revealed a thick exudative process in the meninges, consisting of two zones. There was an inner zone of lymphocytic and fibroblastic infiltration immediately overlying the cerebellar cortex. The outer zone consisted of masses of polymorphonuclear leucocytes, and of many large mononuclear phagocytes. A vertical section through the third ventricle just behind the optic chiasm showed considerable organization of the basilar exudate with the formation of many small vascular channels and the proliferation of numerous fibroblasts. Sections through the brain abscess at the tip of the temporal lobe showed a sinus filled with many degenerating polymorphonuclear leucocytes, communicating the meningeal exudate at the base of the brain, just to the left of the optic chiasm with the abscess cavity. The abscess cavity was filled with a mass of necrotic polymorphonuclear leucocytes, among which were observed three or four dark blue-staining masses with the poorly defined fibers radiating from the periphery. These bodies were suggestive of actinomycotic colonies. The surrounding brain tissue contained many small blood vessels and many fibroblasts, but there was no definite abscess wall. Sections through the cortex, remote from the abscess, showed engorgement of the meningeal vessels and slight disseminated areas of rarefaction of parenchyma. In the brain stem there were small areas of rarefaction, particularly in the parenchyma, subjacent to the thick meningitic process. An occasional lymphocytic perivascular infiltration was encountered.

Bacterial stains failed to demonstrate the presence of bacteria in the meninges or in the brain abscess. The small dark-staining bodies described above appeared in the bacterial stain as dark, homogeneous purplish bodies in which no rod-like structures could be distinguished. The radiating, fibrillary processes were gram negative. These bodies morphologically resembled the ray fungus of actinomycosis.

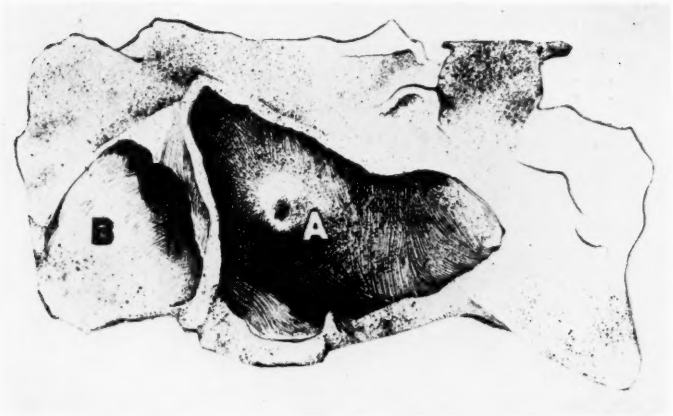

Fig. 3. Coronal section through both sphenoidal sinuses. (A) The summit-like prominence on the posterior wall of the left sphenoid. (B) Small right shallow sphenoid situated anterior to well pneumatized left sphenoid.

Paranasal Sinuses.—The ethmoid and sphenoid paranasal sinuses with the adherent dura were removed en bloc. Grossly, there was no apparent defect in the posterior wall of the sphenoid or any visible communication between the sphenoid sinus and the cranial cavity. The dura over the sphenoid was considerably thickened and firmly bound to the basisphenoid. The surface was covered with yellow purulent exudate. The right ethmoid labyrinth presented the status post intranasal spheno-ethmoidectomy with complete exenteration of the ethmoid cells. The corresponding sphenoid was widely open and contained a small amount of thick mucopus. The left middle turbinate and sphenoid were not disturbed, but on probing the left sphenoid natural orifice some greenish mucopus was obtained. The block was preserved in formalin and then treated with 5 per cent nitric acid. When completely decalcified, the block was divided in the midline at right angles to the cribriform plate, thus exposing both sphenoid sinuses. A localized prominent swelling could be seen on the posterior wall of the left sphenoid, producing a summit-like elevation of mucosa. The diameter of this raised area was about 1 cm. At the apex of the swelling a dimple in the mucosa could be seen. The appearance was that of a furuncle surrounded by an area of infiltration (Fig. 3). A marked discrepancy in the relative size of the sphenoid sinuses was now very evident. The right sphenoid was much smaller than the left and situated more anteriorly. The left sphenoid was

Fig. 4. Photomicrograph of horizontal section through the posterior wall of left sphenoid at the level of prominence A. in Fig. 3. (Low power.) (A) Actinomycotic colonies surrounded by a compact zone of polymorphonuclear leucocytes. (B) Osteomyelitis of the posterior wall of sphenoid extending endocranially.

extensively pneumatized, protruded behind the right sphenoid and almost encircled it. The block was now prepared routinely for serial sections and the slides stained with hematoxylin and eosin after celloidin imbedding.

Microscopic examination revealed an actinomycotic sphenoid osteomyelitis and meningitis. The furuncle on the posterior wall of the left sphenoid consisted of a subepithelial actinomycotic abscess containing numerous colonies of ray fungi. The mucosa of both sinuses was edematous. The ray fungus was intimately imbedded in a compact layer of degenerated polymorphonuclear leucocytes, forming a circular abscess. Surrounding this area there was an acute inflammation of the mucosa with invasion by polymorphonuclear leucocytes and occasional lymphocytes. The marrow spaces were obliterated by an infiltration of inflammatory cells and fungi. Areas of distinct bone destruction with polymorphonuclear invasion of the cortex and multinuclear giant cells (osteoclasts) could be seen. The pathway of infection, endocranially, could be traced by the extension of the osteomyelitis to the overlying meninges (Fig. 4). The dura was

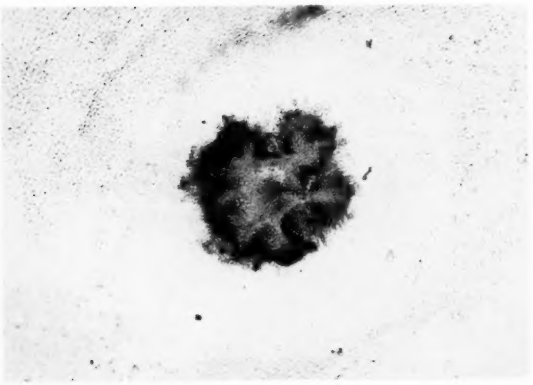

Fig. 5. Photomicrograph of an actinomycotic colony from the meninges. (Medium power.)

markedly thickened and infiltrated with inflammatory cells. Numerous actinomycotic colonies were present in the dura, covering the basi sphenoid (Fig. 5). The dura investing the third, fourth and sixth nerves in the right sphenoid fissure contained numerous colonies of fungi. The posterior bony wall of the right sphenoid was the seat of a similar actinomycotic osteomyelitic process. Small areas of brain substance (pituitary) showed no inflammation. There were no thrombi in the larger vessels. The ray fungi colonies were definitely identified as actinomycosis.

Comment.—The method of introduction of the actinomyces into the body is uncertain. Because of the frequency of this infection amongst farmers and laborers, many observers believe that the infection is exogenous. They contend that the disease is acquired either by picking the teeth with straws or chewing grass or hay. Lord[5] obtained actinomyces from carious teeth and tonsillar crypts, and reproduced the actinomycotic tumors in the guinea pig by intraperitoneal injection. He maintains that the spread of disease in the body is from within, during a period of lowered resistance or following trauma.

It should be emphasized that while this case is reported as primary actinomycosis of the sphenoid, this diagnosis was entertained only after complete postmortem examination failed to reveal any other possible focus. The mode of entry into the sphenoid is still not clear.

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CII.

SYPHILITIC TONSILLITIS: HISTOPATHOLOGY IN THE SECONDARY STAGE.*

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It is apparent from a review of the medical literature that the opportunity for histologic study of syphilitic tonsillitis in the secondary stage of the infection is rare indeed. It has been demonstrated clinically, however, that the tonsils are commonly involved in this phase of syphilis. In 1910 Campbell[1] found that spirochaeta pallida could be found in films from the tonsils in 80 to 90 per cent of cases of secondary syphilis. This period extended from the time of general glandular enlargement until the end of active secondary signs. In a small number of his cases the ease of finding spirochetes in tonsillar films proved a useful method of diagnosis. He observed that enlarged and hyperemic tonsils invariably revealed spirochetes; enlarged tonsils without hyperemia yielded spirochetes with a great degree of certainty; and in most cases of secondary syphilis where the tonsils appeared normal spirochetes were found. It is therefore apparent from Campbell's observation that the tonsils are almost always involved in secondary syphilis.

Much of the literature on syphilis of the tonsils is devoted to primary and tertiary involvement. This report of two cases that are definitely in the secondary stage affords a study of the lesion of secondary syphilis of the tonsil and confirms the observation made by Campbell. Both of these patients exhibited a secondary rash, one at the time of and the other soon after tonsillectomy. One of the patients had been treated in the outpatient department of the University Hospital for an ulcer of the lip, probably the primary sore. In the other patient no primary sore was discovered by her physician, nor was one observed by the patient. Because of her sex the primary lesion could have passed unnoticed by the patient, and had probably healed before the time of the general physical examination ten days

*From the Department of Pathology, University of Georgia School of Medicine. Read at the meeting of the Richmond County Medical Society, June 20, 1935.

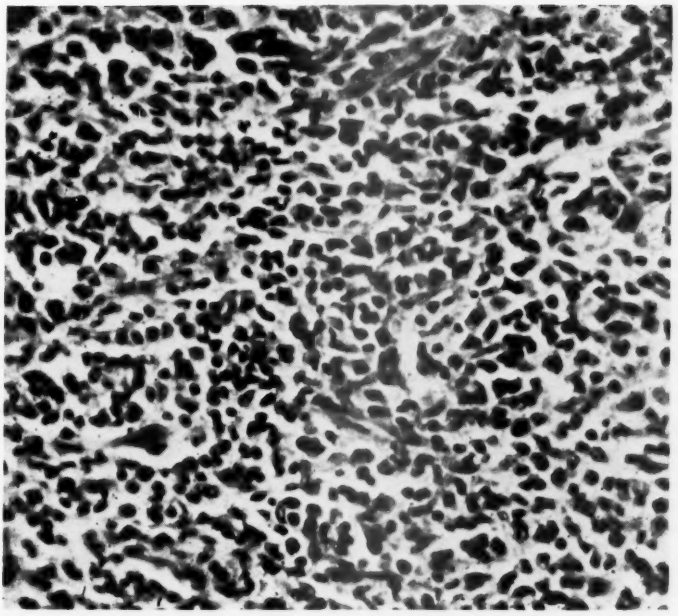

Fig. 1, Case 1. Diffuse fibroblastic and angioblastic proliferation in the lymphoid stroma. Large mononuclear cells, fibroblasts and new grown capillaries have replaced much of the lymphoid tissue. Hematoxylin and eosin. X 500.

after tonsillectomy. The diagnosis of syphilitic tonsillitis in each case was made from the histologic study and the demonstration of spirochetes, which were characteristic of the spirochaeta pallida. The diagnosis in each instance was subsequently confirmed by the Wassermann reaction on the blood of the patients. It is not our custom to make routine sections of tonsils that are received in the laboratory. The peculiar gross appearance of the tonsils of case 1, warranted further histologic study. Later with this gross picture in mind, the tonsils of case 2, because of their similarity to the former pair, were suspected of being syphilitic and this suspicion was confirmed by the histologic study.

REPORT OF CASES.

CASE 1.—C. S., a mulatto female, aged thirty-two, complained of a constant sore throat for two months. The tonsils were large and congested. Tonsillectomy was advised and performed. The pathologic diagnosis was syphilitic tonsillitis. One week after operation a maculopapillary rash appeared on the neck, arms and upper part of the body. At this time the Wassermann reaction on the blood was strongly positive. The rash disappeared after the administration of neo-arsphenamin.

Fig. 2, Case 1. Spirochaeta pallida are seen in the stroma. Silver impregnation. X 720.

The tonsils were considerably enlarged and possessed a narrow base, each tonsil measuring 1 x 2.5 x 2.5 cm. The surface was almost smooth, indented by only a few crypts. The cut surface was pale, slightly fibrous and the number of crypts was apparently diminished. On microscopic examination the tonsils were diffusely altered from the normal. The number of lymphocytes was diminished and the follicles were small and few in number without germinal centers. Much of the lymphoid tissue was replaced by large mononuclear cells, fibroblasts and a new growth of capillaries. (See Fig. 1.) This change involved the lymphoid stroma and gradually encroached upon the lymph follicles. The large mononuclear cells resembled those that are derived from reticuloendothelium. There were a few indistinct foci where the monocytes resembled epithelioid cells, and at these sites an occasional giant cell was observed. The giant cells were similar to those seen in tuberculosis. This picture suggested Hodgkin's disease without the presence of Reed-Sternberg cells. However, when sections were stained by the method of Dieterle,[2] the diagnosis was obvious because of the presence of numerous spirochetes with the characteristics of the spirochaeta pallida. (See Fig. 2.) The spirochetes were found in the lymphoid stroma but not in the remains of the follicles. They were especially numerous along the new grown capillaries.

Case 2.—D. C., a white male, aged nineteen, entered the hospital for tonsillectomy because of recurrent attacks of sore throat for several years. For the past three weeks he had had a coryza and recently he had complained of mild muscular pains. Seven weeks previously he was treated in the outpatient department for a sore on the upper lip which had now healed. The tonsils were inflamed and enlarged. A small acuminate pustular eruption was noted over the entire body. The leukocyte count was 12,200 and temperature was 99.5°. For two days after ton-

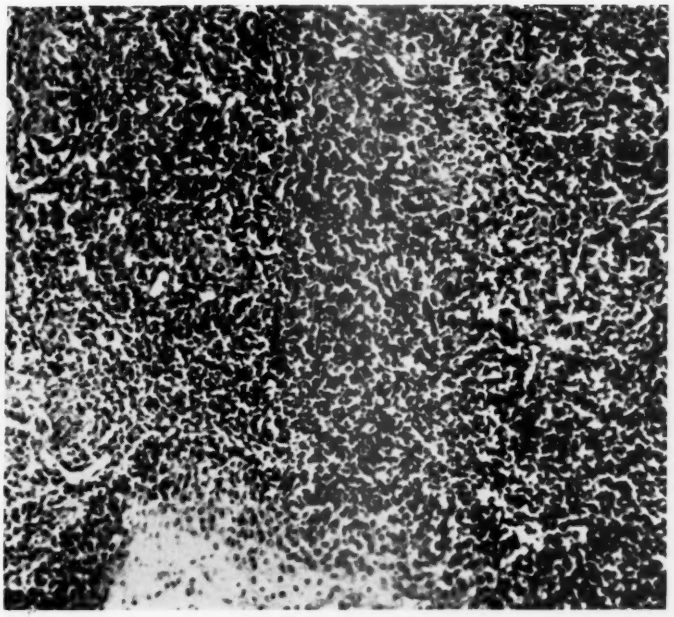

Fig. 3, Case 2. Accompanying the diffuse reaction are foci of epithelioid cells. These are seen in the vicinity of the epithelial lined crypts. Hematoxylin and eosin. X 135.

sillectomy his temperature varied between 98 and 102 degrees. On his own responsibility he left the hospital with a temperature of 101°. The pathologic diagnosis was syphilitic tonsillitis. He was dismissed from the hospital before the completion of the histologic study and was temporarily lost sight of. Nine months later he attended the outpatient department because of a penile sore of only two weeks' duration. There were two irregularly oval ulcers one-quarter inch in diameter on the penis, and dark field examination of a film from the lesions revealed the spirochaeta pallida. The Wassermann reaction on the blood was strongly positive. When informed of the previous diagnosis, Dr. J. W. Brittingham, in charge of the department of syphilis, thought the penile lesion was a recurrent secondary reaction.

The tonsils were large and in the gross strikingly resembled those of case 1, but microscopically they differed slightly. Many large lymph follicles were present, most of them containing large germinal centers. The lymphoid stroma, however, was altered by diffuse fibroblastic activity, a new growth of capillaries, and the production of large mononuclear cells of the reticuloendothelial type. The number of lymphocytes of the stroma was reduced. In minute areas the monocytes resembled epithelioid cells and here an occasional multinucleated giant cell was seen. These foci, while irregularly scattered, were more numerous just beneath the covering epithelium of the surface and of the crypts. (See Fig. 3.) With Dieterle's[2]

stain, spirochaeta pallida were demonstrated. The organisms were scattered throughout the lymphoid stroma and were found in large numbers in the foci of epithelioid cells.

From these foci there was an apparent streaming of the spirochetes towards and into the surface epithelium. In the capsule lymphocytic infiltration occurred about the tubular structures.

COMMENT.

Syphilis of the tonsil has been noted particularly in the late stage of the disease and as a primary lesion. The pathologic picture of the tonsils from these two patients differs from most of those described, and the occurrence of the lesion at the time of a secondary rash indicates that the lesion is early. Weller,[3] in an examination of 8,697 tonsils, stated that the differential diagnosis between tuberculosis and syphilis depended on the relatively avascular character of the former and the arrangement of the epithelioid cells in whorls about minute capillaries in the latter. This whorling effect was not noted in our specimens. One case of syphilis of the tonsils was observed by Morris[4] in a study of 2,321 tonsils, and he described areas of epithelioid tissue in the deeper parts of both tonsils. Starry[5] found four cases of syphilis in 3,854 pairs of tonsils. One case was congenital in a child, and the other three acquired syphilis in adults. He concluded that the latter three represented at least a relatively late stage of acquired syphilis. In the congenital case areas of epithelioid proliferation were scattered throughout the section and there were also foci of fibroblastic and angioblastic proliferation with polymorphonuclear leucocytic infiltration. Two of the acquired cases showed areas of marked epithelioid proliferation scattered throughout the sections and these foci were especially numerous in the germ centers of the lymphoid tissue. He, too, noted in the former two cases epithelioid cells in whorls. Thus Weller, Morris and Starry have directed particular attention to the focal lesions which are probably characteristic of late and hereditary syphilis. The third case reported by Starry, which presented a histologic picture similar to that of our cases, was probably a manifestation of secondary syphilis. The tonsils of this case were moderately enlarged, and practically all of the lymphoid tissue was replaced by marked epithelioid proliferation and occasionally a portion of a germ center still remained.

Apart from those syphilitic lesions of the tonsils as manifested by the primary chancre, mucus patches and an ulcerative pharyngitis, a distinctive tonsillitis occurs in both the hereditary and the acquired type of the disease. In acquired syphilis the tonsillitis may be a secondary or a tertiary manifestation. In the tonsillitis of the

hereditary type and of the tertiary stage of the acquired type focal lesions are characteristic. While in the secondary stage of the acquired type the tonsillitis is characterized by a diffuse reaction in the lymphatic stroma which is described in the report of these two cases.

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CIII.

THE SORE THROAT IN EARLY SYPHILIS.*

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For the past eight years, during observations of patients with early syphilis I have been more or less impressed by the frequency of the subjective complaints of sore throat. Since January, 1931, I have examined and questioned 1,226 patients with various lesions of early syphilis at the Syphilis Division of the Out-patient Department of the University of Georgia Clinic. This number of patients comprises the group seen at this clinic from January, 1931, until June 1, 1935. They were classified further as follows: 423 patients with primary syphilis, 502 with typical secondary manifestations, and 301 with so-called early syphilis. The latter group was made up of patients whose disease was assumed to have been one year or less in duration, but in whom there were none of the usual signs of primary or secondary syphilis. The majority of these patients gave a history of having had a chancre or a generalized rash at some time within one year of their admission to this clinic, but at the time of admission presented a wide variety of complaints, such as vague joint or muscle aches, "misery in the chest," fainting spells, etc. Many of them had no definite complaints, but had been referred on account of discovery of a positive serologic reaction during routine examinations. It is in this group particularly that the frequency of sore throat proved to be of considerable interest.

Before reviewing the records of these 1,226 patients, I discussed the frequency of sore throats in early syphilis with several laryngologists. The result of these discussions led me to believe that this was indeed a rather common manifestation of early syphilis, but when I attempted to review the literature regarding this subject I was surprised to find that it had been given little consideration. There were many descriptions of tonsillitis and other throat lesions in tertiary syphilis, and occasional references to primary syphilis in this region, but very little mention has been made to this type of pathology during secondary or so-called early syphilis. As long ago as 1910 Campbell[1] reported his observation that spirochaeta pallida could be demonstrated in films from the tonsils in 80 to 90 per cent of patients

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with secondary syphilis. It is apparent that Campbell's work was either not repeated on a large enough scale or not sufficiently emphasized. The object of this report is to add clinical emphasis to Campbell's very much earlier observation, in the hope that more careful histories will be obtained from, and more frequent or routine serologic studies will be made in young adults who might appear with the type of sore throat and tonsillitis to be described.

Of the patients with primary syphilis 47 out of 423, or 11.1 per cent, complained of sore throats at the time of admission or at some recent time before admission. Most of these patients had had a primary lesion for about two weeks or longer, and some of them would probably be better classified as very early secondary syphilis. This classification was not considered justifiable, however, because of the absence of pathognomonic secondary lesions such as mucous patches and skin rashes. The appearance of the tonsils in this group was very much like that in those with early secondary lesions and will be described later. The small number of sore throats here is mentioned chiefly by way of comparison with the larger percentage in the other two groups. It is well known that general lymph-glandular enlargement is a rather common sign of early syphilis, and since the tonsils are lymph glands, it is not difficult to understand why they are not involved in the very first days of syphilitic infection.

A study was made of 502 patients with typical secondary syphilis, and out of this group 125, or 24.9 per cent, gave histories of sore throats, either on admission or shortly beforehand. In most of these patients the tonsils were enlarged, and presented dirty ulcerations. In many of them, however, the tonsils were smoothly and cleanly enlarged, with very little evidence of other inflammation, such as redness. In the former group secondary infection was obviously present, and it would be impossible to differentiate such tonsils from those infected with streptococcus or the mixed throat infections which are so frequently seen in patients without syphilis. The absence of constitutional symptoms, such as fever, general malaise, etc., is quite conspicuous in the patient with a syphilitic throat, and might make one suspicious in adult patients. In the very early cases a helpful sign is unilateral cervical glandular enlargement of a discrete character—that is, glands which are enlarged separately and more or less in chains. When felt with the gloved finger these tonsils which are free of ulceration are found to be rather hard and firm. The consistency of the ulcerated tonsils is definitely less firm and is sometimes spongy in character. Stokes[2] has said that a sore throat of more than one week's duration in the presence of a macular rash is almost always due to syphilis.

Of the group of patients with so-called early syphilis a still higher incidence of tonsillitis and sore throat was found. One hundred and seven, or 35.5 per cent, out of 301 of these patients showed very definite throat or tonsil disease. The glands in this group were more or less like those in the secondary group in that they appeared out of proportion to the absence of constitutional symptoms. The sore throat of which this group complained was usually of more than one week's duration.

In regard to diagnosis, it would be a relatively simple matter to do dark field examinations of films from suspected tonsils, but such a procedure was not resorted to in this study because all of these patients had strongly positive Wassermann and Kahn tests on their sera at the time of admission. The percentage of positive serologic tests for syphilis is always at its highest level at this stage of the disease, regardless of the type of test performed. In a crowded clinic dark-field examinations are not usually done when the diagnosis has been established by previous recent serologic tests. Further aid to diagnosis of this type of disease is the response to treatment, since syphilitic tonsils usually appear normal after three or four arsphenamin injections.

CONCLUSION.

Sore throat and tonsillitis in early syphilis are relatively common symptoms of this disease.

Subjective complaints of sore throat and objective evidence of tonsil disease were found in 28.8 per cent of 803 patients with secondary and early syphilis.

One hundred and twenty-five out of 502, or 24.9 per cent, of patients with secondary syphilis had evidence of tonsil abnormality.

One hundred and seven out of 301, or 35.5 per cent, of patients just beyond the secondary stage presented signs of tonsil disease.

Routine serologic tests for syphilis should be performed on all adult patients with sore throat of more than one week's duration, particularly if constitutional symptoms are absent.

Grateful acknowledgment is made to Mrs. Ethel Bruker, R. N., and the Social Service Department of the University Hospital for assisting me in summarizing these cases.

DOCTORS' BUILDING.

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CIV.

CONGENITAL FIBROEPITHELIAL CYST OF THE NASAL VESTIBULE: REVIEW OF THEORIES OF PATHOGENESIS.*

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CHICAGO.

Many names have been applied to cystic tumors found on the floor of the nose. These include serous or mucous retention cyst, alar cyst, mucoid cyst, soft tissue cyst and cyst of the anterior nares, the floor, the lateral wall or of the vestibule of the nose. This varied terminology based on location or contents of the growths, disregarding microscopic structure, is indicative of the earlier state of uncertainty as to their nature or origin.

In 1892, Zuckerkandl[1] made the first brief mention of a cyst projecting into the nasal passage, at the base of the alar cartilage. Early reports were contributed by Chatellier, McBride, Dunn, Milligan and Knapp. (2) Brüggeman[2] collected fifty-two cases in the literature up to 1920. In an article containing a good bibliography Arnoldi[4] listed twenty cases in the period up to 1929. Additional reports to date bring the total up to some eighty-odd cases. Except for mention in St. Clair Thomson's textbook[5], no report in the English literature of the past decade has come to my attention.

The following case corresponds with typical descriptions.

REPORT OF A CASE.

CASE 1.—E. R., a white female, 16 years old, was seen on September 9, 1932, with a fullness of the left cheek at the alar angle, obliterating the nasolabial groove. This swelling had been gradually increasing over a period of six years. Nasal breathing was partially obstructed on that side. There was no pain. Within the nose could be seen a hemispherical swelling, elevating the normal mucosa of the floor and distending the lateral wall of the vestibule. The mass was soft and elastic on palpation. The inferior turbinate was displaced medially and its lower surface was concave due to pressure. Some fullness could be felt in the gingivolabial sulcus, but there was no bulging or softening of the hard palate or superior maxilla.

No changes indicative of polypoid or suppurative disease of the nose or accessory sinuses were apparent on examination. The maxillary sinuses transmitted light

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with equal brilliance. The teeth were in good condition. There were no other relevant physical findings. The Wassermann reaction of the blood was negative. The diagnosis made was: Cyst of the lateral wall of the nose; type undetermined.

Roentgenograms of the sinuses indicated only a slight haziness at the periphery of the medial wall of the left antrum. There was no evidence of supernumerary teeth or of bone changes in the superior maxilla.

Aspiration of the fluctuant swelling within the vestibule yielded four cubic centimeters of a thin, honey colored, translucent fluid. With the needle in situ an equal amount of iodized poppy seed oil 40 per cent was injected into the cyst cavity. Anteroposterior and lateral roentgenograms were taken with the head vertical. Immediately thereafter the antrum was entered by trocar inserted through the lateral wall of the inferior meatus, well behind the cyst. Nothing could be obtained on aspiration and irrigation of the cavity. Seven c. c. of iodized oil was then instilled into the sinus and the roentgenograms repeated in the previous positions.

In the anteroposterior and lateral roentgenograms the cyst was visualized lying on the anterior wall of the antrum, encroaching into the pyriform aperture. No opaque oil can be seen in the sinus cavity. In the second series of x-rays the antrum was seen to be partially filled with the iodized oil with no filling defects to indicate abnormal changes of the mucous membrane. The absence of communication between the cyst and the sinus cavity is clearly shown by the intact wall of the superior maxilla.

The fluid aspirated from the cyst contained amorphous debris and epithelial cells. Cholesterol was not present.

Under local anesthesia, a sublabial incision was made exposing the cyst lying in the incisor fossa on the anterior surface of the maxilla, distending the membranous portion of the lateral nasal wall. There was no connection with the teeth or bone, but sharp dissection was required to separate the sac from its intimate union with the nasal mucosa. The nasal cavity was not entered. The cyst contained a glairy mucopus, evidently due to secondary infection at the time of aspiration a few days preceding the operation. The incision healed promptly; a moderate postoperative reaction of the cheek subsided in a few days. Inspection one year later showed a normal floor and lateral wall of the nose. There was no facial asymmetry.

The removed cyst measured 20 by 15 by 5 mm. On microscopic examination the wall of the sac consisted of a fibrous connective tissue stroma containing areas of hyalinization, many fibroblasts, young capillaries and blood spaces. Some areas were hemorrhagic; others infiltrated with numerous leucocytes. There was no definite connective tissue basement membrane. Glandular elements were absent. The lining was a stratified columnar nonciliated epithelium, containing goblet cells and some basal nuclei. Although cilia were absent, the epithelium corresponded to that of the upper respiratory tract.

On the basis of microscopic appearance, Eckert-Moebius[6] has used the term fibro-epithelial cyst to describe similar formations.

SYMPTOMATOLOGY.

Cysts of the nasal vestibule are usually unilateral; however, several bilateral cases have been observed.[7] Kasche noted similar cysts in a father and daughter.[8]

It is unexplainable that the incidence is almost exclusively limited to the female sex. Occurrence in the male was reported in only one of the fifty-two cases collected by Brüggeman.

The rate of growth is usually slowly progressive. With increasing size of the cyst, partial obstruction to nasal breathing and asymmetry of the face result. Pain is absent unless infection follows trauma or aspiration. Spontaneous fistulæ do not occur. Slight pressure atrophy of the inferior turbinate or superior maxilla is not infrequent.

Effective treatment requires complete operative excision. Aspiration of the contents of these cysts is often followed by recurrence, despite attempts at chemical cauterization.

DIAGNOSIS.

In the differential diagnosis, benign and malignant tumors of the nose, including carcinoma, sarcoma, papilloma, lymphangioma and the various chronic granulomata, including those of syphilis, lupus, tuberculosis and others, must be considered. These lesions may usually be distinguished by their relative firmness and obvious changes in the nasal mucosa.

In mucocele of the maxillary sinus the lateral nasal wall may be distended and elastic but not fluctuant. There may also be thinning of the hard palate or antrum wall, with "parchment-like" crackling on pressure. Transillumination shows greater brilliance than the opposite side. Glairy mucoid contents are obtained on aspiration or irrigation.

Dental cysts may be confused with vestibular cysts. They are either of follicular or radicular origin and include simple cysts, dentigerous cysts, odontomas and adamantinomas. These formations usually arise within the body of the alveolus or maxilla and contain rudimentary or complex tooth elements. Roentgenograms often show a connection with unerupted teeth, apical granulomas or other bone changes. The microscopic picture is variable, but squamous or modified squamous epithelium is the rule. Cholesterol is frequently found in the cyst contents.

REVIEW OF THEORIES OF PATHOGENESIS.

There has been considerable controversy regarding the genesis of cysts of the nasal vestibule. Earlier writers maintained that they were retention cysts originating in the mucous glands of the nasal mucous membrane. Brown-Kelly[9] supported this contention by histologic demonstration of numerous glands in the vestibule. He assumed that cysts arose from glands the ducts of which had been blocked by inflammatory changes. This theory was refuted in view of the lack of connection between the cysts and the nasal mucosa.

On the basis of their location at the root of the nasal ala, Zarnico[10] ascribed the origin of vestibular cysts to perichondritis of the cartilage. This assumption has been disproved by the absence of inflammatory or retrogressive changes in the alar cartilages.

Later writers considered these cysts to be congenital malformations, but differed concerning the structure from which they might originate. Grosser[11] suggested the possibility of derivation from a vestigial remnant, the glandula lateralis nasalis (Steno's gland), which, in animals, is located opposite the maxilloturbinal. Finding a rudimentary form of this gland in a human embryo, he speculated as to its potentiality for later cystic maldevelopment. Uffenorde[12] denied this possibility by showing that the theoretical location of this gland, homologous to its position in animals, would be opposite the agger nasi in the human nose and thus remote from the vestibule. Further, such vestigial remnants have not been found in postfetal existence in man.

Grünwald[13] considered a persisting remnant of the nasopalatine duct to be the origin of nasal cysts. Confirming this possibility, Schaeffer[14] writes as follows:

"Approximately two cm. dorsal to the inner margin of the nostril and in juxtaposition to the nasal septum, each nasal fossa presents a slight depression in its floor. This depression leads into a small canal lined with mucosa . . . the nasopalatine canal (canalis incisivus, canal of Stenson) courses obliquely caudalward and with its fellow of the opposite fossa converges toward the nasal septum, descends almost vertically and passes through the Y-shaped incisive foramen (anterior palatine canal) in the hard palate. The nasopalatine canals are remnants of the wide communication between the nasal and oral cavities found at an early period of fetal life. In the vast majority of instances, the lumina of the canals are obliterated and represented by impervious cords of epithelial cells . . . The obliteration of the lumina often begins before birth. The persistence of the lumen of a portion of the nasopalatine canal may be the explanation for cells in the maxilla dorsal to the upper incisor teeth. Indeed, such a cell could communicate either with the inferior meatus or the buccal cavity; or in the event that both extremities of the canal become impervious, the central portion would be without a drainage channel and could readily become cystic."

Cysts arising from this source should be found on the medial side of the floor of the nasal passage and thus at a distance from the constant location of true vestibular cysts, namely, beneath the root of the ala. Schaeffer cites one instance of such a cyst in the former unusual position.

Levin[15] concurred with Wingrave[16] in regard to the possibility of cystic formation from the vomeronasal organ. He further indicated the connection of this vestige with the rudimentary nasopalatine duct. Cysts arising from Jacobson's organ would, therefore, be found on the nasal septum or the medial side of the nasal floor.

Impressed by the location of the majority of cysts near the lateral nasal wall, Brüggeman advanced a theory of origin from a degenerated portion of the nasolacrimal duct. In embryologic studies of the development of this duct in animals, Peter[17] indicated the variable position of its orifice in the inferior meatus. In those cases in which the opening of the duct occurred proximal to the extreme end, the portion distal to the orifice regressed. Growth of this vestige was inferred as an explanation of the cysts. Cysts from this source would more likely occur in the floor of the nose than in the lateral wall.

The hypothesis with which most of the recent authors agree ascribes the genesis of vestibular cysts to developmental faults in the closure of the primary facial processes. In the course of fusion of these structures there may occur inclusion of islands of ectoderm. Such epithelial rests, buried by closure of clefts, remain dormant unless some undetermined stimulus in postnatal life causes resumption of growth.

A description of the formation of the nose and face is essential to a comprehension of this mechanism of embryologic maldevelopment. According to Schaeffer,[18] in the fourth week of fetal life the nasal pits become separated by the primitive frontonasal processes. This differentiates into the lateral nasal process (which later forms the alæ nasi and lateral wall of the nose) and the medial nasal process (which later forms the septum and central part of the upper lip). At this time, also, the mandibular arch gives rise to the maxillary processes, which later form the upper jaw, the cheeks and the lateral part of the upper lip. About the thirty-fifth day, obliteration of the primary facial clefts occurs through fusion of the various facial processes. "For a brief time the lines of fusion of the maxillary and lateral nasal processes with the median nasal processes are represented by strands of ectodermal tissue. These ectodermal fusion lines soon disappear. . . . Persistent epithelial cell masses may later in extrauterine life give rise to epithelial pearls and cysts."

Blumenthal[19] emphasized the observation of Brown-Kelly that the location of lateral nasal wall cysts is at the fusion point of the embryonic facial processes.

Klestadt's reports[20] firmly established the concept of fissural genesis of vestibular cysts, which he termed "gesichtsspaltencysten," or "facial cleft cysts." In a consideration of all possible origins of seromucous cysts, his complete classification includes:

I. Typical Forms.

1. True vestibular cysts: located in the lateral wall of the vestibule at the pyriform aperture. These are definitely of facial cleft origin.
2. Secondary vestibular cysts: located in the floor of the vestibule. May arise variously from:

(a) Facial cleft inclusion rest.
(b) Nasopalatine duct vestige.
(c) Nasolacrimal duct vestige.

II. Atypical Forms.

May theoretically arise from any portion of the nasal mucosa and be due to:

1. Congenital malformations of the mucous glands.
2. Embryonal growths of floor of nose.
3. Inflammatory retention cysts.

Klestadt concludes that the great majority of vestibular cysts are of facial cleft (fissural) origin and may be included in types I, 1, and I, 2a. He concedes that a minority, depending on their location, may arise from other embryonic vestiges or from inflammation. These conclusions have been accepted by most of the recent authors.[21]

COMMENT.

It is a cardinal principle that congenital malformations usually arise at points where fusion of embryonal layers favors inclusion of fetal rests. Resumption of growth later results in formations characterized by the original growth potentiality of the vestige, in this instance a mucous cyst arising from the anlage of a portion of upper respiratory tract mucosa.

In explanation of the pathogenesis of most vestibular cysts, the fissural rest concept may be considered most tenable in view of the following facts:

First, the constant location of these cysts at a site of predilection for fetal rests, namely, the junction point of three primary facial processes, corresponding anatomically to the root of the nasal ala.

Second, the histologic picture of such cysts, showing epithelium of the upper respiratory type, and,

Third, the occurrence of bilateral and familial cases.

No single name hitherto employed correlates the genesis, structure and anatomic location of true vestibular cysts. For this reason the combined descriptive term of "congenital fibro-epithelial cyst" of the nasal vestibule is suggested.

SUMMARY.

1. A case is reported of a congenital fibro-epithelial cyst of the lateral wall of the nasal vestibule. This is a true vestibular cyst.

2. In a review of theories of genesis, the embryologic origin is generally accepted.

3. Following Klestadt's classification, a minority of vestibular cysts may arise from vestiges of the nasolacrimal or nasopalatine ducts; the origin of the majority, however, may be explained by the fissural genesis concept, which relates their derivation to epithelial rests buried by inclusion during the closure of the primary facial processes in embryonic development.

NOTE.—Since this paper was completed an article by Laszlo[22] has appeared reporting three cases of "So-called Mucoid Cysts of the Nose." Case 1 has the usual characteristics of a vestibular cyst. However, the microscopic descriptions in Cases 2 and 3 mention bone formation and areas of stratified squamous epithelium. Also, cholesterol was found in the contents of these cysts. In my opinion these findings make it advisable to consider the latter cases with reservation.

As to the origin, the author supports the theory that . . . "they develop either from the epithelial sheet of Hertwig or from misplaced enamel germs mixed with misplaced embryologic sinus germs . . . ;" and further . . . "all cystic growths of epithelial character which are located around the alveolar process and even in the whole maxillary process are nothing else but dentigerous cysts of probably follicular type and develop from the paradental epithelial cells."

It is conceivable that these statements may explain some cases, such as numbers 2 and 3 in the author's report. One cannot be positive regarding theories of embryonic malformations. However, considering the strong evidence in favor of the fissural genesis concept, it is difficult to accept the theory of dentigerous origin of vestibular cysts.

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Symposium
Before the American Otological Society, Toronto, May 28, 1935.

SUBJECT: CERTAIN FUNDAMENTALS IN REGARD TO SUPPURATION OF THE PETROSAL PYRAMID.

I. Introductory Remarks ..... Samuel J. Kopetzky, New York City

II. Normal and Pathologic Anatomy of the Petrous Pyramid.

(1) Stacy R. Guild, Ph. D. ..... Baltimore
(2) Marvin F. Jones, M. D. ..... New York City
(3) J. Gordon Wilson, M. D. ..... Chicago
(4) Edmund Prince Fowler, Jr., M. D. ..... New York City

III. The Clinical Picture and Diagnosis of the Various Types of Infection.

(1) Ernest M. Seydell, M. D. ..... Wichita, Kans.
(2) Curtis C. Eves, M. D. ..... Philadelphia
(3) C. Stewart Nash, M. D. ..... Rochester, N. Y.

IV. The Fundamentals of Therapy.

(1) John R. Page, M. D. ..... New York City
(2) William V. Mullin, M. D. ..... Cleveland
(3) Isidore Friesner, M. D., and J. D. Druss, M. D., New York City
(4) Ralph Almour, M. D. ..... New York City
(5) Wells P. Eagleton, M. D. ..... Newark, N. J.

V. Summation and Conclusions.

(1) H. I. Lillie, M. D. ..... Rochester, Minn.

VI. Discussion.

(1) S. J. Kopetzky, M. D.
(2) Harold G. Tobey, M. D.
(3) George M. Coates, M. D.
(4) H. H. Vail, M. D.
(5) F. L. Lederer, M. D.
(6) F. B. Woodward, M. D.

I. INTRODUCTORY REMARKS.

CV.

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This symposium is held in an endeavor to clarify many of the mooted points in regard to infections of the petrosal pyramid.

In opening the discussion, it is necessary to state the problems which confront us. All are agreed that among the lesions which are found in the petrosal portion of the temporal bone, tuberculosis and syphilis are pathologic entities not germane to our present discussion. Likewise, the various types and groups of labyrinthine lesions do not come into the range of our discussion today, except perhaps incidentally.

We are concerned with the tissue reactions to bacterial invasion, by the streptococcus group usually and by the staphylococcus group occasionally, which result in lesions in the petrous portion of the temporal bone. These lesions give rise to symptoms that make diagnosis possible. We are concerned with the endocranial advance of these lesions, which may terminate in death from meningitis unless spontaneous arrest of progression occurs and healing results without the application of any of the special therapeutic measures devised specifically for the arrest of the lesion.

Bacterial invasion of the petrosal portion of the temporal bone results in a lesion whose pathologic entity is determined by the character of bone structure in which the infection locates itself. Dependent upon this, there is found either an osteomyelitis or a coalescent osteitis.

These two distinct types of bone lesions have totally differing mechanics of evolution and different aspects on microscopic examination. They give different clinical pictures and, by and large, occur in differently aged individuals. Failure to recognize these essential points is one of the prime factors which has brought about so much discussion and varying opinions among us.

We know that osteomyelitis occurs in bone which has bone marrow—hematopoietic and fatty marrow—and that osteitis can only occur in bone which has undergone pneumatization. In our early discussions, therefore, it was natural enough for Gordon Wilson

to report on microscopic studies on young children's bones and question me on the presence of pneumatization in such. Subsequent study has shown that we can assume pneumatization when the trabeculated bone spaces are lined with a delicate membrane like that in other air containing spaces; and assume in its absence that in the process of preparation for microscopic study fatty marrow may have been dissolved out, leaving cavities which are artefacts, and which erroneously may have been regarded as evidence of pneumatization. The time after death at which fixation is made influences the preservation of this lining membrane—the sooner after death, the better the microscopic picture. But the presence or absence of some remaining bone-marrow-filled spaces is not enough to entirely clarify the mooted question at issue.

It is necessary to recall another fundamental. The development of the labyrinthine capsule and its contents is much further advanced at birth than is the rest of the temporal bone, and at this time its visceral and cortical surfaces are separated from each other by a series of trabeculæ in whose meshes there is bone marrow. We know from the studies of Wittmaack how pneumatization proceeds. Normally about the fourth year of life pneumatization is usually completed in the mastoid process. During these early years of life pneumatization also progresses in the petrosal portion of the temporal bone. No one has yet firmly established at what age normal pneumatization becomes completed in this section of the temporal bone. What is known is that there is a large degree of variation in location and extent of pneumatization, and that a certain admixture of marrow containing and pneumatized types of bone occurs in the given specimen. The younger the individual examined, the more true this becomes. Nor can this mixture of standard types do more than give a predilection to the form which the lesion will present in the given case. When a coalescent osteitis develops in a partially pneumatized petrosa, with small remains of marrow carrying trabeculæ, the infection will either halt when it reaches the unpneumatized parts, as Eagleton believes, or it will invade vascular channels and give rise to osteomyelitis. Autopsy reports have given us ample proof of pus pockets in the basal parts of the pyramid adjacent to the epitympanum and antrum, and at the same time sequestra formation in the deeper parts, anterior to the labyrinthine capsule.

In infants and very young children, therefore, we should expect the absence of pneumatization and the presence of hematopoietic and fatty marrow in the bony trabeculæ. In young adults, it is reasonable to expect a degree of pneumatization which roughly runs parallel

to the degree of pneumatization found in the mastoid process. There is a clinical means of differentiation between the two types, which we shall not now discuss. Since the roentgen films have been in general use to study bone structure in an infected temporal bone, we have at hand a certain means to estimate the character and the extent of pneumatization which the patient presents. We can, therefore, predicate both the kind and the nature of the lesion which is the basis of the pathologic process. Comprehended, this clarifies another moot question. In infants this method of diagnosis is obviously unnecessary.

If one reviews the literature reporting autopsies, from 1858 to the present time, another finding becomes evident. Osteomyelitic lesions of the petrosa are more often bilateral than unilateral, and the presence of these lesions is more frequently noted in very young children and infants than among adults.

Compact bone—the so-called sclerotic bone—does not take part in either of these two types of lesions. Yet, as a matter of fact, it is to be noted that some elements of diploic bone—the bone which carries bone marrow—always persist as "rests" in compact bone. (The term "compact bone" is understood here as a descriptive term of macroscopically studied bone structure.) Under the microscope, diploic "rests" will usually be demonstrable; yet this type of bone evidences resistance to ordinary bacterial infection. I know of no reports of its decalcification or coalescence; nor have I seen records of its involvement in osteomyelitic processes. I have known of sequestra formation due to trophic and nutritional disturbances. However, these do not concern us in this discussion.

Finally, when considering these osteomyelitic lesions of the petrous portion of the temporal bone, it is well to remember that those who have specially studied this lesion divide it into two types: (A) Hematogenic and (B) otogenic. A differential diagnosis has been worked out by Ramadier.[1]

Since we are today stressing diagnosis, let it suffice to point out that in the hematogenic type of osteomyelitic lesion, the general symptoms dominate the clinical picture, and that the tympanic and otomastoidal signs are relatively insignificant. It is usually bilateral. General and even pulmonary phenomena develop step-for-step as the diploe becomes progressively infiltrated, until meningitic reactions supervene and death ensues. Ramadier, Renaud[2] and others who have studied this lesion state: "They have no tendency toward spontaneous cure. Unoperated, they always advance more or less swiftly toward death. Fortunately this lesion is relatively rare."

On the other hand, otogenic osteomyelitis develops significant middle ear suppuration. This lesion became established as a pathologic entity in 1888 by Jaymes,[3] in 1893 by Pauzat,[4] Mignon[5] in 1898, Broeckaert[6] in 1899, G. Laurens[7] in 1902, Luc[8] in 1905 and 1910, and Guisez[9] in 1906.

It was Luc who stressed the rôle which the venous system of the bones plays in the evolution of this lesion. Regression phlebitis spreads the intra-osseous infection, facilitates its advance and eventually carries it to the endocranium (G. Laurens-Durand[10]).

The otogenic type of osteomyelitis occurs after the acute phase of the otitic infection has subsided. At the time of its special development the tympanic infection is usually subacute or verging on chronicity (chronicity here being a protracted tympanic suppuration). But the onset of the osteomyelitic lesion is signalized by acute signs of sudden violent auricular and peri-auricular or hemicranial pains. There is noted simultaneously an increase in the intensity of the otitic suppuration.

It seems strange that in all the discussions that have been held in our societies, with the possible exception of Eagleton, none have taken this lesion into account. All have written and spoken as if the pathologic lesion in all petrosæ were identical and similar; and the relative value of therapeutic measures, both surgical and non-surgical, have been considered without any attempt at a clear comprehension of the underlying differing pathology. Obviously, identical surgical therapeutics are inapplicable to differing lesions.

I desire to pay tribute to Eagleton for his outstanding point of view on osteomyelitis of the petrosa. His studies were neither as extensive nor as ordered as the French authorities' (see above), yet he realized the significance of this lesion as few of us had.

In my own contributions,[11] in collaboration with Almour, our first studies, published in 1930, definitely stated that we confined our report specifically to osteitis of the petrosal pyramid. We endeavored to fix the significant characteristics of the clinical picture and advocated a method of surgical attack adapted to one type only of encapsulated coalescent osteitis.

A large and very rich literature is in existence dealing with the osteitic type of bone lesion. The pathologic entity was well established in the early part of this century. The mechanics of its evolution were comprehended and the manner in which nature evacuated the purulency was well known. The autopsy reports of many

of the cases dying with a terminal meningitis record fistulous tracts leading into the depths of the pyramid.

Again, sometimes at operation a surgeon was led beyond the usually exposed area of the mastoid cavity to follow purulent tracts whose openings he had inadvertently discovered. Often these tracts were discovered at secondary operations for revision of the mastoid surgery, and the result was crowned with varying success (see Wesley C. Bowers, Laryngoscope, June, 1928).

By and large, it might be said that the clinicians, not comprehending the relationship of their findings to the symptomatology, nor understanding the sequence of events until the well known clinical picture of meningitis supervened, were unable to set diagnostic data and establish standards for surgical indications to relieve the lesion.

To add to the confusion, a misconception ensued regarding the significance of the Gradenigo syndrome. Because so many patients came to autopsy whose terminal illness presented the syndrome and whose postmortem examination showed purulent involvement of the deepest part of the petrosal pyramid, it became the thought of the day that the Gradenigo syndrome was significant of a lesion at or near the tip. This view gained currency notwithstanding the opposition of authorities like Panse,[12] Boonacker[13] and Huizinga, Geronzi,[14] Körner,[15] Alt,[16] Köller,[17] Lange,[18] Uffenorde[19] and Vogel,[20] and in this country, Sears,[21] Eagleton,[22] Kopetzky[11] and Almour.[11] I believe that rational progress in understanding the clinical manifestations which reveal themselves in coalescing osteitis of the petrosal portion of the temporal bone was unduly impeded by this persistent misunderstanding.

Another fundamental requires consideration here. Unlike the mastoid process where pneumatization is uninterrupted and the cells are intercommunicating, the pneumatization of the petrosal portion of the temporal bone seemingly runs in tracts which irregularly and varyingly fuse with one another. Almost all of them merge at the so-called tip. In the earlier stages of an infection in these tracts, the lesion is more marked in one or another of them. If recognized early enough, the infection may be found isolated and not necessarily involving all of the tracts.

It has become habitual, therefore, for those observers who know this to consider coalescent osteitis of the petrosa as being:

1. Posterosuperior
2. Posteroinferior

3. Posterointernal
4. Anterior supracochlear
5. Anterior subcochlear
6. Anterior precochlear (pericarotid)
7. Empyema of the apex cells.

Roughly, this locates the tracts leading from the tympanic and epitympanic areas forward and inward around the labyrinthine capsule, toward the so-called apical termination. I am using the nomenclature suggested by Almour.

Dependent upon the time when the given lesion is recognized clinically, another subdivision is possible and a further differentiation of clinical signs is feasible to add to diagnostic acumen. One should designate a given lesion as (A) *intrapetrous*, on the one hand, and (B) *peripetrous* on the other. The intrapetrous lesion may be diagnosed from evidence it presents before endocranial extension; the peripetrous lesion from the clinical manifestations of involvement of the various surrounding tissues—nerves, blood vessels and muscle stratæ.

Finally, one more point needs presentation for discussion. Many cases which give symptoms denoting an involvement of the petrosal bone get well with no further surgery than a well performed simple mastoidectomy. Others, when carefully studied, show symptoms which lead to the discovery of a fistulous tract which is reachable without resort to radical mastoidectomy. When the discovered tract is curetted and widened to enhance drainage, these too usually recover. Then there are cases in which the tract is located at the tubotympanic orifice, which can only be reached after a radical mastoidectomy has been performed. Finally, there are the encapsulated empyemas, with their threat of intracranial invasion, which require specialized technics in order to be reached and evacuated.

In determining which form of surgical therapy to apply, the term "conservative treatment," as opposed to surgical attack, or "radical treatment," should have no place. I am only commencing to comprehend the dangers inherent in the so-called "conservative treatment." Any measure, regardless of its nature, which fails to reach and evacuate the lesion is hardly "conservative." It should more correctly be designated as "futile."

On the other hand, radical mastoidectomy and the application of specialized technics are unnecessary, and must not be employed as "routine" measures in all types of cases. Should we not rather suit our therapy to the problem which the given case presents, and

term that form of therapy "conservative" which brings relief and recovery in the shortest and most direct way? Nor is it necessary to operate on all cases. Careful study of the factors presented, repeated roentgenograms and a knowledge of how the lesion progresses will soon give the observer that experience which is necessary to appreciate and recognize those cases which will recover without additional surgical attack. If such studies were made more often, reported cures of cases which, to say the least, are, upon critical analysis, regarded as "diagnostically equivocal," would appear less frequently in the literature.

With these fundamentals outlined and comprehended, the questions before us today resolve themselves into the following:

1. Can osteitis be differentiated clinically from osteomyelitis of the petrous bones? Upon what shall diagnosis depend? What rôle shall roentgen examinations hold in the determination of diagnostic factors?

2. Is the triad of pain from the first branch of the Vidian nerve, continued otorrhea, or a reappearance of otorrhea after a period of cessation, accompanied by a low grade sepsis, common to all types of petrositis? If not, to what particular types does this triad of symptoms apply?

3. Do lesions in different portions of the petrous bone give characteristic symptoms of sufficient import to enable a localization of the site of the lesion preoperatively?

4. Do we need a more exact nomenclature to describe the lesions in the petrous bone so that advocated technics may be better understood?

5. What shall be understood by "conservative treatment"?

6. Since it is possible to diagnose the lesion during the stage designated "intra-petrosal," is it considered "conservative" or proper to institute surgical measures which attack the lesion through peri-petrosal areas?

7. Since there are obviously types of petrosal involvement and varying localizations of the site of the lesion, are all types and all sites amenable to one type of therapy? Let us specify the type and location of the site of the lesion to which a given therapeutic measure is applicable.

8. At what time in the development of the lesion does surgery become necessary?

9. What are the hearing and other end results in proven cases of petrosal infection when only simple mastoidectomy is performed?

It is my hope that, in the ensuing exchange of experiences and viewpoints, we shall be able, in the calm of philosophic reasoning, to progress toward the solution of these problems.

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II. NORMAL AND PATHOLOGICAL ANATOMY OF THE PETROUS PYRAMID.*

CVI.

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From the standpoint of descriptive gross anatomy the outstanding feature of the human petrous pyramid (pars petrosa of the temporal bone) is its extremely wide range of "normal" variation. This variation concerns not only size and external configuration but also the nature and arrangement of the internal structure. Its range of normal variation is even greater than that for the mastoid region. Some petrous pyramids have extensive, complete pneumatization—the cells may be large, small or medium sized; other petrous pyramids have no pneumatization beyond the limits of what are commonly regarded as middle ear and eustachian tube; and every possible intermediate degree of pneumatization occurs.

The structure of nonpneumatized pyramids and of the nonpneumatized portions of partially pneumatized pyramids is also variable. Any region may consist of cancellous (spongy) bone with the spaces almost entirely occupied by hematopoietic (red) marrow or by an almost pure fatty marrow or by a mixture of these two types of marrow, or it may consist of solid, dense bone with only a very small amount of marrow. The petrous pyramid is frequently made up of a mixture of pneumatized spaces, solid bone, cancellous bone with red marrow and with fatty marrow; there is no regularity to the arrangement of these tissues with reference to each other.

The external form of the petrous pyramid is so variable that there is not even agreement between the writers of anatomic and oto-

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logic textbooks as to the number of surfaces, and angles between surfaces: by the English, American, German and French authors the pyramid is described as having four sides and angles about as frequently as having three sides and angles (exclusive of base and apex). And yet I feel certain that each author described accurately the temporal bone he was looking at when he wrote. The fact is, the petrous pyramid of the human temporal bone does have a very wide range of "normal" variation.

In textbook accounts even the descriptive terms used to indicate directions differ: the anterior angle of some authors is the lateral angle of others, etc. The difference in the textbook descriptions is, I believe, responsible in large part for the confused ideas that are prevalent with respect to the anatomy, the pathology and the surgery of the so-called "petrous apex."

This leads us to the brief consideration of another principal source of confusion: the term "petrous apex." If there ever was a misnomer from a descriptive standpoint, this term is one of the worst. Mathematically considered (that is, from the viewpoint of solid geometry), the pars petrosa of the temporal bone, whether it have three or four sides, is a truncated pyramid and does not have an apex, but only a smaller end. In the anatomic texts (and the anatomic portions of most otologic texts) the term apex applies only to the irregular *surface* of the smaller end of the pyramid. In the clinical and surgical literature—and in our daily conversation—the term "petrous apex" is used in two very different senses, neither of which means the same as the anatomic term. Many clinicians speak of the apex as that portion of the petrous pyramid located anteromedial to the cochlear part of the otic capsule (Fig. 1). At the same time, from the practical standpoint of operative surgery many have come to think of, and to speak about, the petrous apex as that part which is not cleaned out during a radical mastoid operation: this definition of the apex includes all of the petrous pyramid anteromedial to the arch of the superior semicircular canal. The very different areas indicated by the term "petrous apex," almost always without definition by the authors using them, have added to the confusion.

There is variation in the structure of the petrous pyramid, there is variation in the descriptive terminology, and the same terms have been used to designate different areas: a frank recognition of these facts is, I believe, the first step in clarifying the problem which is the topic of the symposium for today.

Fig. 1. A semi-schematic representation of the ear and its position in the skull as seen from above. (Modified from Eckert-Möbius, in the Denker-Kahler Hdbch. d. H.-, N.- und Ohrenheilk., Bd. VI.) All of the temporal bone sections illustrated in this account were cut approximately parallel to the plane indicated by the line marked "vertikale Schnittrichtung." Figs. 3 to 15 are from sections through the region of the horizontal part of the carotid artery; Figs. 16 to 18 are from sections through the ascending part of the carotid artery and the anteromedial part of the basal turn of the cochlea; and Figs. 19 and 20 are from sections through the perilabyrinthine region. Except for Figs. 3 and 16 all sections are from the temporal bones of adult individuals.

I shall not attempt to say who is right and who is wrong, or which term is the better or which term is misleading. Any term may be used without causing confusion so long as everyone understands what is meant by the term.

With the exception of the otic capsule and its contents there is no bone or bone marrow, epithelial, fibrous or other tissue in the petrous portion of the temporal bone that is peculiar to this region of the body. It will suffice, therefore, to give but a very brief description of each major structural element.

The framework of the petrous pyramid, exclusive of the otic capsule, usually consists of so-called cancellous (or spongy) bone with a cortical or surfacing layer of varying thickness. This cancellous bone has irregular trabeculæ and septa which form the boundaries of irregularly shaped spaces, just as in other parts of the body where cancellous bone is normally present.

The irregular spaces in the cancellous bone of the petrous pyramid are filled with one or the other of four things: hematopoietic (red) marrow, fatty marrow, a mixture of red and fatty marrow, or air-containing cavities.

Histologically, the marrows in these spaces are indistinguishable from the corresponding types of marrow in other bones of the body. This is true whether the marrow be present in large or in small amounts.

The walls of the air containing cavities of the petrous pyramid are indistinguishable histologically from those of the mastoid region. In both regions the pneumatic cells (or pneumatized spaces), when present and normal, have a very thin mucosal lining (or mucoperiosteum) that consists of a scantily vascularized fibrous layer covered by a simple squamous epithelium (i. e., a single layer of flattened cells). See Fig. 2. Also, in both the mastoid process and the petrous pyramid, when there is only a partial pneumatization, this thin mucosa sometimes rests directly upon marrow-containing spaces, though usually a thin layer of bone separates air-containing spaces from marrow.

The small regional vessels of the bone also supply and drain the marrow and the mucosa of the pneumatic cells. Nothing is known about the lymphatics of this region. Definite secretory or sensory nerves to the mucosa of the pneumatized spaces have never been demonstrated for either pyramidal or mastoid regions; vasomotor nerves have been described accompanying the blood vessels.

The inner ear—membranous labyrinth, otic capsule, etc.—is of course the central structure of the entire petromastoid portion of the temporal bone: this is the structure around which all of the other parts are developed. Since it is the central or "core" structure, and is at the same time the least variable, a descriptive terminology based upon relations to the otic capsule is more logical than any other, and less apt to be misunderstood. The term "perilabyrinthine" designates a region in a manner that cannot be misunderstood. Certainly the term is neither confusing nor ambiguous. Dr. Marvin F. Jones recently suggested to me the term "*ante*labyrinthine" for that portion of the petrous pyramid anteromedial to the cochlear capsule. This term also is not ambiguous. Since the long axis of the petrous pyramid makes an angle of about 45 degrees with the sagittal plane of the head (Fig. 1), the term "mediolabyrinthine" is also possible. My choice of "antelabyrinthine" is mostly an arbitrary one—the word is

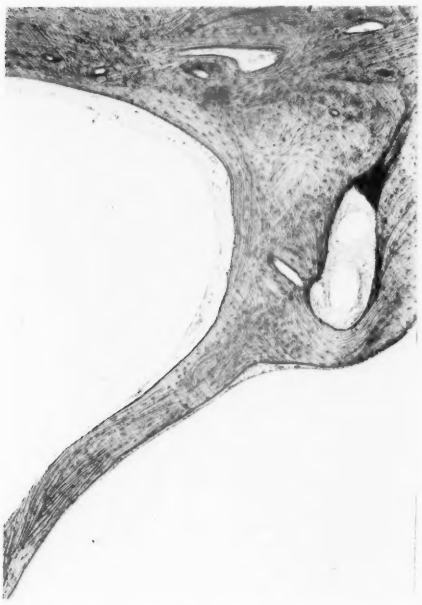


Fig. 2. From the same section as Fig. 3. This magnification shows that the characteristic appearance of the lining membrane of the normal pneumatized spaces of the petrous pyramid is the same as in normal mastoid cells.

easier to say. The region designated by the term "antelabyrinthine" might also be described as the anteromedial portion of the perilabyrinthine region.

The region indicated by the term "antelabyrinthine portion of the petrous pyramid" corresponds to one of the clinical usages of the term "petrous apex," and the combination of "perilabyrinthine" and "antelabyrinthine" portions corresponds to the other clinical usage of the term "petrous apex."

I might interpolate here that, on the same basis, the mastoid region of the temporal bone could be termed the "post-labyrinthine" part of the petro-mastoid complex. And from the standpoint of clear thinking with respect to the anatomy, histology, pathology and surgery of the temporal bone this terminology might be of assistance.

The regions of the human temporal bone first pneumatized are the eustachian tube, the middle ear and the mastoid antrum. Devel-

opmentally each is an extension of the other, in the order named. Pneumatization of the mastoid process takes place by extensions from the mastoid antrum, and the cells of the mastoid region are commonly designated according to their location with reference to other structures: thus, jugular cells, tip cells, zygomatic cells, sinus cells, etc. These terms are clearly descriptive and cause no confusion.

While pneumatization of the mastoid region is always by an extension of cells from but one of the primary areas (with the occasional exception of cells along the jugular region from the hypotympanic part of the middle ear), pneumatization of the perilabyrinthine and antelabyrinthine portions of the petrous pyramid may and does occur by the extension of cells from all three of the primary areas; and indeed, from two regions of the middle ear, from any part of the osseous portion of the eustachian tube, and from any part of the mastoid antrum that is near the otic capsule.

On the basis of their developmental origin (and this is very important surgically) the pneumatic cells that may be present in the perilabyrinthine and antelabyrinthine regions of the petrous pyramid are logically subdivided into four major groups and designated as "tubal," "hypotympanic," "epitympanic" and "antral."

From each of these four regions the pneumatized cellular extensions may, and sometimes do, extend into any region of the so-called "petrous apex"; and further, the extensions may be by several routes from each place of origin. Accordingly the location of a pneumatic cell with reference to some near by landmark is not an infallible indication of the place of origin of the cell (and therefore its place of natural drainage in case of infection). I have even seen cells in the region between the carotid canal and the eustachian tube which, when traced through serial sections, were found to have come from the mastoid antrum along the superior angle, over the internal meatus and then beneath the carotid canal. And in the reverse direction I have followed cells of tubal origin above the cochlea and the internal meatus, also forward into the extreme tip of the petrous pyramid.

Therefore it is logical to designate the pneumatic cells of the petrous pyramid, whether peri- or ante-labyrinthine, by names that are descriptive of their location. When and if the origin of any given cell in an individual case is determined, this information can be added to the designation of location by a phrase. Thus: supracarotid cell from antrum, supracarotid cell from tube, infracochlear cell from hypotympanum, superior-angle cell from epitympanum, supra-meatal cell from antrum, infratubal cell from hypotympanum,

carotico-tubal cell from tube, etc. A complete list of all the cells that sometimes occur would amount to a list of the names of all structures in the region of the petrous pyramid, with prefixes indicating directions. Any name for a cell is good that calls up a definite mental image of its location.

More important than names is to have clearly in mind that pneumatic cells in the corresponding regions of different petrous pyramids may orginate from regions as far apart as mastoid antrum and eustachian tube. From the clinical viewpoint it is also very important to have clearly in mind that even cells in the same location and from the same origin differ widely not only in the size of their terminal expansions but also in the size of the connections that lead back to their point of origin. For instance, one supracarotid cell may be small and separated from the contents of the carotid canal by a thin lamina of bone, but separated from the dura mater of the middle cranial fossa by marrow-containing cancellous bone and by a thick layer of cortical bone; while another supracarotid cell may be several millimeters in diameter and separated from both the carotid canal contents and from the dura by thin laminæ of bone (Fig. 14). And either the large or the small cell may have a wide channel all the way to its place of origin, or the communicating channel from either cell may have a constricted portion. So long as infection does not occur these variations are of academic interest only, and I will not cite further examples or attempt to give details of dimensions or of connecting routes for the several regions. The number of variations is enormous: as yet I have not seen exact duplicates among the temporal bones examined.

In nonpneumatized pyramids there are also variations in the supracarotid and in other regions. Thus there may be either small or large marrow-containing spaces above the carotid canal, or there may be no marrow above the horizontal part of the carotid canal, the upper wall of which then becomes an integral part of the cortical bone of the floor of the middle cranial fossa (Fig. 9). Also, the distance between the ascending part of the carotid canal and the anterior part of the cochlear capsule is exceedingly variable, depending upon the presence or absence of pneumatic cells or of marrow in this particular location (Figs. 16, 17, 18). Wide variations also exist in the distance between the carotid canal and the bony part of the eustachian tube: extremes are illustrated in Figs. 3 and 10. The carotid canal has been selected for these illustrative examples of variations in relations because it is the largest structure in the antelabyrinthine region of the pyramid.

Excellent examples of variations in pneumatization of the petrous pyramid were published by Bezold[1] in 1882 and by Siebenmann[2] in 1897. Their illustrations of preparations made by filling all cavities with melted wax or with Wood's metal and then corroding away the soft tissues and the bone, are still the best in the literature. It is my opinion that were these works better known much of the present confusion with respect to the anatomy of the petrous pyramid would never have arisen.

A large number of functionally important structures either pass through or are in contact with one part or another of the petrous pyramid: the list of names is very long as compared to a similar list for the mastoid region. Description of these relations has been left to the end of this account in order to be able to make the consideration of them more than just a dry recital of descriptive anatomic terms.

Even before Gradenigo published the account of the syndrome that bears his name clinicians diagnosed pathologic states of the petrous pyramid by observing the symptoms produced by involvement of neighboring structures. But, before proceeding further, please permit me to say, as an anatomist who is reasonably conversant with otologic literature and clinical problems, that I do not believe anyone has yet, in making a diagnostic study, taken full advantage of all of the anatomic relations of the petrous pyramid. By a complete appraisal of all positive and all negative clinical observations that can be made, it should be possible to localize the pathologic area. In essence the diagnostic procedure required would be very similar to that used in localizing lesions of the central nervous system: What functions are disturbed; what functions are not disturbed; what are the pathways concerned with these functions and in what regions do all of the affected pathways pass near enough to each other to be involved by a common lesion, etc.? The use of an exactly analogous method of systematic examination and reasoning, based on a thorough knowledge of the anatomy and function of the parts concerned, should yield equally valuable information with respect to the location of pathologic processes in the petrous pyramid.

From this viewpoint of applied anatomy, therefore, the structures that are in relation to the petrous pyramid will be considered. And because it is impossible in a talk to demonstrate clearly the

1. Bezold, F.: Die Corrosions-Anatomie des Ohres. München., 1882.

2. Siebenmann, F.: Mittelohr und Labyrinth. In the von Bardeleben Hdbch. d. Anat. d. Menschen., Bd. 5, Abt. 2. Jena, 1897.

exact relations, and since good descriptions can be found in textbooks and studied at leisure, the accounts of most of the relations will be limited to very brief statements.

The petrous pyramid, besides being continuous with (or articulating with) the other parts of the temporal bone (mastoid, squamous and tympanic), also articulates with the body of the occipital bone and with the posterior angle of the greater wing of the sphenoid bone, and is separated from the body of the sphenoid by the contents of the foramen lacerum. These relations are well shown in anatomic texts, so we need not dwell upon them now, even though the facts are important.

More than half of the total surface of the petrous pyramid is covered by dura mater; this is about evenly divided between that of the middle and that of the posterior cranial fossa. The other layers of the meninges, the cerebrospinal fluid and certain parts of the brain and brain-stem are of course in close relation to the petrous pyramid, and the tentorium cerebelli is attached to its superior angle.

The relations of the petrous pyramid to the middle ear cavity have already been considered, so far as they are of importance for this symposium.

Inferiorly the petrous pyramid has certain relations of possible clinical importance. In the first place, it is very near to the lateral part of the vault of the nasopharynx. The relationship makes it reasonable to suppose that inflammatory reactions in this part of the pyramid might produce local reactions in the vault of the pharynx: so far as I know the nasopharynx is not routinely examined from this viewpoint in patients with infections of the petrous pyramid. Secondly, the tensor veli palatini and the levator palatini muscles take origin in part from the under surface of the pyramid near the tip (Figs. 11, 15). So far as I am aware no one has noted symptoms produced by irritation of these muscles.

The trunks, or the roots, of seven cranial nerves, fifth to eleventh, inclusive, are in close relation to one or another part of the petrous pyramid.

The roots of the trigeminal nerve cross the superior angle a few millimeters from the tip and lie in a shallow groove near the end of the anterosuperior surface of the pyramid; they are separated from the surface of the bone only by the periosteal layer of the dura mater.

The abducens nerve, in its intradural canal, is in contact with the last few millimeters of the end of the posteromedial surface of

the pyramid and, just before entering the cavernous sinus, crosses over the extreme tip of the superior angle.

The symptoms produced by involvement of these two nerves, the fifth and the sixth, are the ones best known: a pathologic process must be near the tip of the pyramid in order to affect either of them.

The facial nerve, besides having contacts with the walls of the internal meatus, is also in close relation with the perilabyrinthine part of the pyramid at the region near the geniculate ganglion (Fig. 19).

The several branches of the eighth nerve enter and have their terminations within the otic capsule.

The glossopharyngeal, vagus and spinal accessory nerves pass through the dura of the anterior part of the jugular foramen. The glossopharyngeal nerve is here in just as close contact with this part of the postero-inferior angle of the petrous pyramid as are the fifth or sixth nerves at the tip; and the vagus and spinal accessory nerves are very near by and also in a constricted channel. An area of inflammatory change limited to this region of the petrous pyramid should cause symptoms primarily of the pharynx, soft palate and larynx—there would not be involvement of trigeminal or abducens nerves. A gross anatomic preparation in our collection has an anatomic arrangement well suited to produce this hypothetical clinical picture. In an otherwise nonpneumatized petrous pyramid there is a small pneumatic cell in that part of the bone near the glossopharyngeal and vagus nerves. By careful probing it can be determined that this cell is an extension from the hypotympanum, also that the cell has a very constricted "neck." In case of infection, with occlusion of its natural drainage route, the logical symptoms expected would have been those associated with disturbed functioning of the near by nerves. And if these symptoms were recognized as possibly due to such a lesion, localization should be easy. The location of this cell is such that the chances of its discovery by x-ray examination would be very slight. Yet the danger of meningitis from an abscess formation in this region would be as great as from one near the tip of the pyramid. In a patient with glossopharyngeal neuralgia and a history of otitis media, an infected cell in this region of the pyramid may be the etiologic factor.

Besides the main stems of seven cranial nerves there are also branches of certain of these nerves and of the sympathetic nervous system that are in close relation to the petrous pyramid.

The greater superficial petrosal nerve, a branch of the facial nerve (or rather, of the so-called nervus intermedius portion of the

facial nerve), is embedded in dura in a groove that passes along the entire length of the anterosuperior surface of the pyramid from the hiatus fallopii to the lateral side of the foramen lacerum. Vail has called attention, in a paper presented before this Society three years ago, to one symptom that may be due to irritation of this nerve: retrobulbar pain. The greater superficial petrosal nerve is one of the large contributors to the sphenopalatine ganglion (via the vidian nerve of the pterygoid canal) and its fibers are concerned with the innervation of all of the large area supplied by that ganglion; therefore an involvement of the greater superficial petrosal nerve should give rise to more manifestations than are yet recognized clinically, such as secretory and vasomotor disorders of the nasal mucous membrane.

The lesser superficial petrosal nerve, a continuation of the ramus tympanicus of the glossopharyngeal nerve (nerve of Jacobson), receives a communicating branch from the geniculate ganglion region of the facial nerve, and in its course passes through the superolateral part of the perilabyrinthine and out along the surface of the antelabyrinthine part of the petrous pyramid, where it occupies a groove parallel to and two or three millimeters anterolateral to that for the greater superficial petrosal nerve. By way of the otic ganglion, the lesser superficial petrosal nerve supplies the parotid gland with secretory fibers (being the so-called cranial part of the sympathetic system for this gland—autonomic or parasympathetic of different terminologies). So far as I am aware, no one has ever sought for unilateral disturbances of parotid gland function in patients with infections of the petrous pyramid.

The chorda tympani branch of the facial nerve, after passing across the middle ear cavity, is in contact with the lateral part of the petrous pyramid as it passes through the Glaserian fissure (petrotympanic suture).

The carotid nerve (or internal carotid sympathetic plexus), which passes through the antelabyrinthine part of the petrous pyramid in the carotid canal, is the direct continuation upwards of the cervical sympathetic trunk. Many of the structures of the head receive the thoracicolumbar portion of their sympathetic innervation by this route. Among other things, one branch of the carotid plexus, the great deep petrosal nerve, is distributed with the branches of the sphenopalatine ganglion. Attention has already been called to the region supplied by this ganglion as offering diagnostic possibilities that have been neglected. The innervation of the dilator muscle fibers

of the pupil is also by nerve fibers that course in the carotid sympathetic plexus. I know of no observations on the relative size of the two pupils in patients with lesions of the petrous pyramid.

Most of the dura mater over the petrous pyramid is supplied with sensory fibers from the meningeal branch of the ophthalmic division of the trigeminal nerve known as the ramus tentorii; filaments diverge to each surface of the pyramid from near the attachment of the tentorium cerebelli. The dura mater near the jugular foramen is supplied by the meningeal branch of the vagus nerve.

To complete the list of nerves in relation to the petrous pyramid, mention should also be made of the nerve to the tensor tympani muscle (which is itself in the superolateral part of the pyramid), the nerve from the pharyngeal plexus to the walls of the eustachian tube (sensory and secretory fibers) and the small communicating branches from the carotid plexus to the nerve of Jacobson.

The blood vessels in relation to the petrous pyramid, besides supplying or draining its tissues, are of importance as affording potential routes for the spread of infectious processes.

The relations of the carotid artery need no further description than has already been given. The communications of the space around this artery, both cranialward and cervicalward, are well known. The venous plexus around the carotid artery communicates freely with the cavernous sinus, which lies just anterior to the tip of the petrous pyramid. The cavernous sinus also communicates freely with the superior and the inferior petrosal sinuses, which in turn communicate with the transverse sinus and the jugular bulb, respectively. The relations of these venous channels are well known.

From the standpoint of our symposium today, the most important thing about all of these venous channels is that they receive small vessels from the parts of the petrous pyramid near them—i. e., the blood from the petrous pyramid is not assembled into one channel before leaving the confines of the bone.

The eustachian tube passes through the anterolateral part of the petrous pyramid; its relations to other structures are somewhat variable, as has already been pointed out. As one of the four regions from which pneumatic cells frequently extend out into the pyramid it is especially important in all considerations of the region.

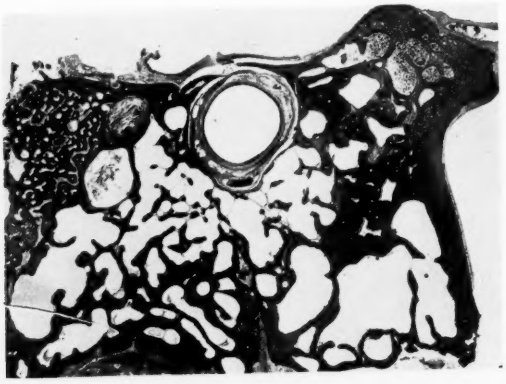
The attempt has been made to present this topic, the anatomy and histology of the petrous pyramid, from a rather different view-

point than is usually taken. I shall not attempt to summarize the facts and suggestions presented. In closing, I only wish to emphasize again (1) that all of the structural elements in the petrous pyramid —in both peri- and ante-labyrinthine portions—are the same as occur elsewhere in the body; (2) that there is an extremely wide range of variation in the arrangements of these structural elements with respect to each other and to the structures which pass through or near the petrous pyramid, and (3) that a thorough knowledge of the variations in the detailed anatomy of the region is essential from the standpoints of pathology, of clinical diagnosis and of surgery.

Fig. 3. Extensive pneumatization of the antelabyrinthine part of the petrous pyramid of a 16-year-old boy. Study of the serial sections of this ear shows that the cell in the superior angle, which in the single section appears to be isolated, is in reality continuous with the main pneumatized cavity. In spite of the otherwise extensive pneumatization of this petrous pyramid, note that there are no pneumatic cells lateral to or above the eustachian tube and the tensor tympani muscle (cf. this region in Fig. 5).

Fig. 4. The spaces of the cancellous bone of the nonpneumatized petrous pyramid of this 51-year-old man are filled with marrow which is predominately of the hematopoietic type.

Fig. 5. In this otherwise extensively pneumatized petrous pyramid there are areas of fatty and of red marrow in the spaces of the cancellous bone medial to the horizontal part of the carotid canal. Note the pneumatized cells lateral to and above the eustachian tube and the tensor tympani muscle. The distance between the carotid artery and the middle meningeal artery (seen in the upper left-hand corner of the picture) is in this section more than twice that in the section illustrated in Fig. 10.



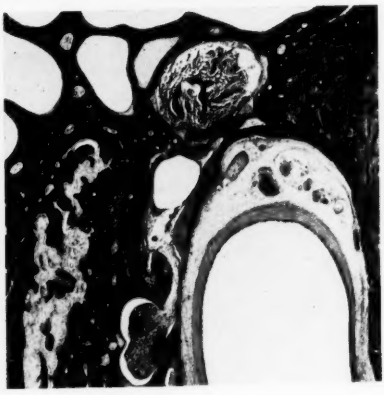
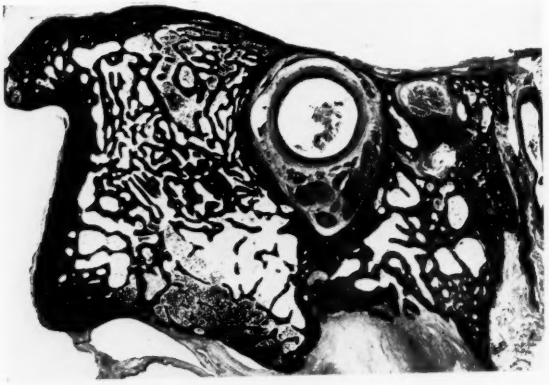
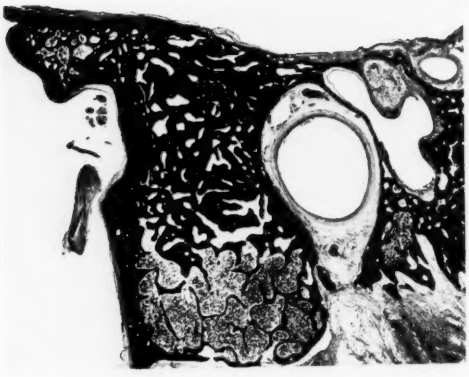


Fig. 6. The spaces of the bone near the superior angle of this partially pneumatized petrous pyramid contain fatty and mixed marrow, and there are thick-walled pneumatized spaces in the region occupied by marrow in the preceding figure. The lining membrane of the large cell below the eustachian tube is in contact, over a small area, with the sutural ligament of the temporosphenoidal articulation. The greater wing of the sphenoid bone is marrow-containing.

Fig. 7. There is a dehiscence of the bony wall of the carotid canal. In case of an infection of the adjacent caroticotubal pneumatic cell an early involvement of the contents of the carotid canal would have been probable.

Fig. 8. Another example of a dehiscence in the bony wall of the carotid canal. The region of the bony defect is in this case in contact with the connection of a small infratubal cell to the eustachian tube, which is very close to the carotid canal in spite of a fair degree of pneumatization of this petrous pyramid. The patient had, at the time of death from pneumonia, an early stage of acute purulent otitis media and beginning involvement of the pneumatic cells of the pyramid.





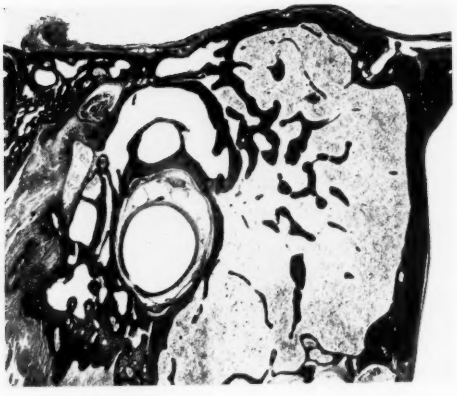


Fig. 9. Most of the marrow in this entirely nonpneumatized petrous pyramid is of the fatty type (cf. Fig. 4). Comparisons of the bony framework of the region medial to the carotid canal in this figure with those seen in Figs. 4, 10 and 11 demonstrate one cause of variations in appearance of the roentgenograms of normal nonpneumatized petrous pyramids.

Fig. 10. The major tissue medial to the carotid canal in this petrous pyramid is bone, the small spaces in which contain fatty marrow. The large spaces inferiorly and the small spaces near the superior angle contain a mixture of red and fatty marrow. The connection of the proximal part of a small supracarotid cell to the eustachian is seen in this section. In the upper right-hand corner of the picture is the middle meningeal artery (cf. Fig. 5).

Fig. 11. In the medial part of this partially pneumatized petrous pyramid the marrow spaces are of the very large type (cf. Figs. 4, 9 and 10). The greater superficial petrosal nerve, coursing in the form of five small bundles of fibers, is in the dura mater over the uppermost of the chain of three supracarotid pneumatic cells. The lesser superficial petrosal nerve is, in this section, in the small bony canal between the more medial two of the chain of small pneumatic cells above the tensor tympani muscle. The levator palatini muscle is attached to the surface of the pyramid near the lowermost of the infratubal cells.

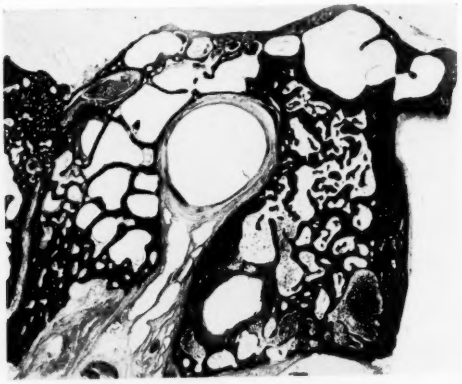


Fig. 12. In this partially pneumatized petrous pyramid there are red-marrow-containing spaces in the bone above the chain of pneumatic cells which extends from the eustachian tube over the carotid canal and out into the superior angle.

Fig. 13. In this partially pneumatized petrous pyramid a large cell extends from the tubal region beneath the carotid canal and into the posteroinferior angle: there is no pneumatization of the red-marrow-containing bone near the superior angle. Infections of the pneumatized cells shown in this and in the preceding figure should give rise to different groups of symptoms.

Fig. 14. This partially pneumatized petrous pyramid has cells in both of the regions that are pneumatized in the pyramids illustrated in Figs. 12 and 13, and a region of marrow-containing bone between the more medial parts of the superior and inferior cells.

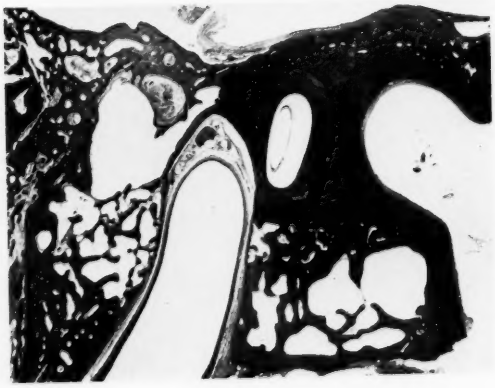
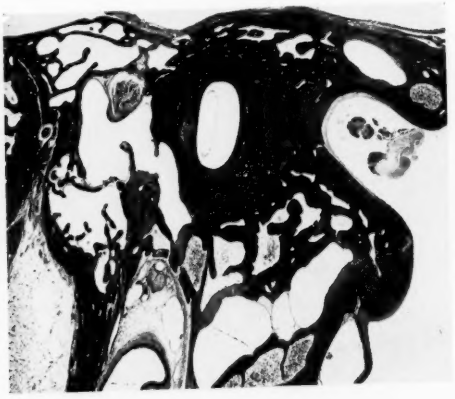


Fig. 15. The serial sections show that the pneumatized space near the superior angle of this pyramid is a cellular extension from the epitympanum (*not* from the mastoid antrum) forward over the internal auditory meatus; also that it is not connected to the chain of carotico-tubal and supracarotid cells, which are in this case of tubal origin. When the pneumatic cells of the pyramid have different origins an operative procedure that successfully drains one group may fail entirely to afford drainage to the other infected cells. Note the fat-marrow-containing space between the pneumatic cells above the carotid canal.

Fig. 16. This section, through the ascending portion of the carotid artery, is from tne same series as is Fig. 3. Even in extensively pneumatized petrous pyramids the carotid artery is frequently in contact with the cochlear part of the otic capsule.

Fig. 17. The relation of the carotid artery to the cochlear capsule is similar to that illustrated in the preceding figure, but there is a great difference in the extent of the pericochlear pneumatization in the two pyramids. This section is from the same series as is Fig. 6.



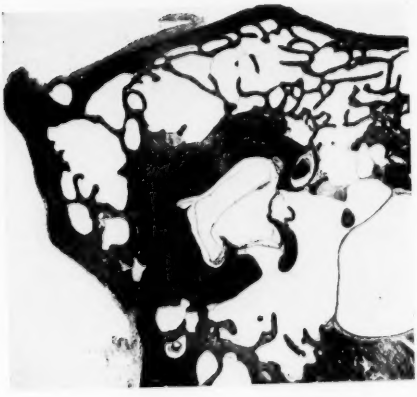


Fig. 18. In this petrous pyramid the carotid artery and the cochlear capsule are separated by pneumatic cells and marrow-containing spaces. This section is from the same series as is Fig. 11. Note that the sections illustrated in Figs. 16, 17 and 18 are all through the same part of the basal turn of the cochlea.

Fig. 19. This petrous pyramid has fairly extensive, but not complete, pneumatization of the pericochlear region: there are small areas of fatty and of red marrow inferiorly. Note the relationship of the geniculate ganglion (just above the middle and apical turns of the cochlea) to the pneumatized spaces.

Fig. 20. This section, through the oval and round windows and just in front of the arch of the superior semicircular canal, shows an extensive degree of pneumatization of the perilabyrinthine region of the petrous pyramid. The bone of this region may, like that of the antelabyrinthine part of the pyramid, have marrow-containing spaces, a mixture of air- and of marrow-containing spaces, or it may consist entirely of solid, dense bone.

CVII.

MARVIN F. JONES, M. D.,*

NEW YORK.

The pathology of the pneumatized pars petrosa of the os temporale presents a few controversial phases. Although disease causes tissue changes in the petrous pyramid which are comparable to the tissue changes in bony structures elsewhere in the body, yet certain features of petrositis resemble no universally recognized pattern. Various theories have been advanced by which to explain unusual tissue arrangements, and many of these theories have been accepted as facts without sufficient corroborative evidence. Recognizing that knowledge of the whole subject of petrositis is in the phase of development, our president has suggested that remarks be limited to personal observations and experience. Reference to former work will therefore be omitted.

More thorough study of the anatomy and pathology of the petrous pyramid should tend to clarify the problems presented by patients with purulent otitis and its complications. Persistent aural discharge accompanied by fever, headache, vertigo, nausea, vomiting, impaired hearing, neuralgia, paralyses, or other symptoms indicating an extension of infection beyond the confines of the petrous cells, creates a puzzling situation.

Dr. Guild has demonstrated the proof of his assertion relative to the analogy between the anatomy of the pars petrosa and the pars mastoidea. Accepting his deductions, it follows as a logical assumption that when the same invading organism involves the anatomic structures he describes, the same pathologic changes will result. The specimens which I have examined (from the collection at the otologic laboratory at Johns Hopkins; Dr. E. P. Fowler, Jr.'s, laboratory at Columbia; Dr. Friesner's laboratory at Mt. Sinai, and those prepared by Dr. Hemsath from my own laboratory at the New York Post-Graduate Medical School) show some proof of this assumption. Those I wish to present are from the collections at Johns Hopkins and Columbia. *They exhibit similar pathologic changes in both the pneumatized petrous and the mastoid portions of the temporal bone.*

*Funds allotted the author by the Central Bureau of Research of the American Otological Society aided in the preparation of this work.

Photomicrographs by Dr. Frederick A. Hemsath.

Dr. Guild has mentioned the difficulties he has encountered with anatomic nomenclature. I have encountered the same difficulties in pathology. To avoid confusion, the nomenclature in common use by otologists will be adopted rather than the more scientifically exact, but to most of us, the less comprehensive nomenclature of the pathologist.

The incidence of pathologic conditions in the pneumatized petrous pyramid has little statistical significance without accurate information regarding the incidence of partial pneumatization. While complete pneumatization is undoubtedly a very rare occurrence, partial pneumatization is not uncommon. Microscopic examination of hundreds of temporal bones is essential before accurate percentage tables can be compiled. My earlier impression was that pneumatization occurs in about one-third of all cases. I recently examined the sixteen adult temporal bones of my collection without finding a pneumatic cell in the antelabyrinthine region of any specimen. Various percentages have been proposed based on gross examination or the roentgenogram. Microscopic examinations prove these estimates to be inaccurate. Only a small percentage of temporal bones have a pneumatized petrous pyramid, and only a small percentage of individuals with a pneumatized petrous pyramid develop petrositis; of these, only an occasional one dies. The temporal bones are obtained at autopsy from only a small proportion of those who die. It is the difficulty in collecting sufficient material for study, therefore, that has retarded the advance of accurate knowledge of the pathologic changes in petrositis.

Nearly all patients with middle ear infection have an accompanying inflammation in the mastoid. The pneumatic cells in the petrous pyramid bear the same or perhaps a more intimate relation to the middle ear cavity than do the mastoid cells, therefore it is reasonable to assume that when the middle ear becomes infected the contiguous petrous cells are also coincidentally involved. Many cases of petrositis probably resolve following the simple operation of myringotomy. By the same reasoning, in patients with a mastoid infection and with cells extending into the petrous pyramid which are structurally similar to and continuous with those of the mastoid region, a petrositis is of necessity an accompanying feature. Obviously, an adequate simple mastoidectomy must, in most cases, effect a resolution of the petrous involvement. It is evident that the pneumatized petrous must be frequently involved, and just as evident that the involvement usually subsides following adequate surgical drainage of the mastoid.

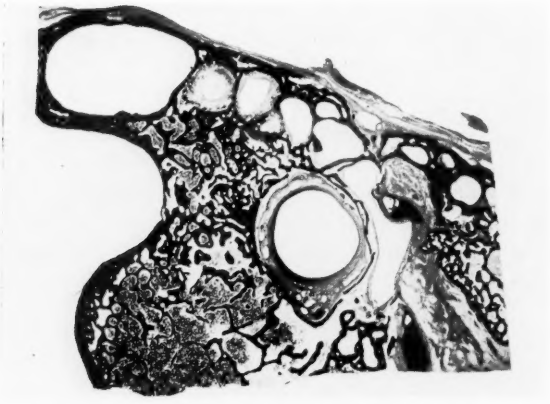


Fig. 1. Case 1.—A vertical section cut through the antelabyrinthine region. This picture shows a pneumatization of the superior cells with one large cell extending along the petrous ridge. The two laterally placed superior cells show a resolved inflammatory process with consequent fibrous organization. The third laterally placed cell has been involved to a lesser degree, which process continues to diminish as the peritubal cells are approached. The supposition is that the original infection traveled by way of the peritubal cells into the superior antelabyrinthine cells where a prolonged resolution occurred.

Chronic infections follow the same general routes as acute involvement. We have in addition the artificial pathways formed by bone destruction. This destruction may involve bony trabeculæ, causing coalescence of pneumatic cells, or may break the confines of the pneumatized spaces and invade the bone marrow. Radical mastoid surgery usually effects drainage sufficient to cure the chronic cases. The type of case I have considered most dangerous is one with an acute infection superimposed upon a chronic or prolonged process. Here we encounter a number of factors which combine to permit the entrance of a new organism into a previously formed cavity, and at the same time obstruct free drainage for the newly formed pus. The original pathway may be obstructed by swelling of the lining membrane, bony and epithelial detritus, fibrous tissue bands or adhesions, and sometimes the consistency of the discharge itself impedes drainage.

The histologic sections reproduced contain evidence to support the above statements.

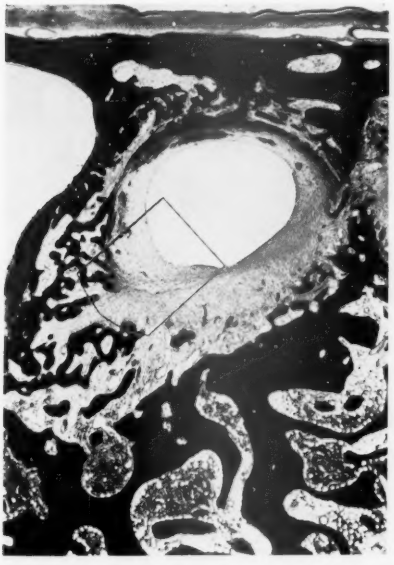


Fig. 2. Case 1.—A higher power showing the extension of an old infection into the marrow spaces, which process would institute an osteomyelitis in addition to the petrous empyema.

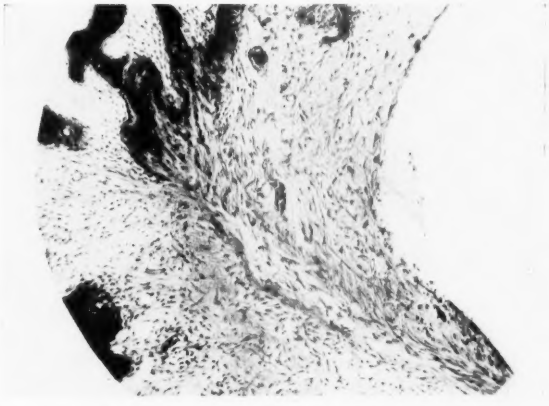


Fig. 3. Case 1.—A detailed picture demonstrating the organized fibrous tissue lining a cell with its extension toward the marrow cells.

Fig. 4. Case 1.—A vertical section at the area of the oval window through the middle ear and tympanic membrane. The structures are normal. The subarcuate cells are shown in this picture, as are also the epitympanic cells which join the superior group and the peritubal cells shown in Fig. 1. An illustration of a case with spontaneous resolution. Death was caused by extra-aural pathology.

Fig. 5. Case 2.—A vertical section at the area of the perpendicular portion of the facial nerve. A cholesteatoma is shown with the squamous epithelium lining the cavity. A group of diseased cells is shown below the cavity which are lined with a modified flat cuboidal epithelium. Judging from the appearance of the area surrounding the cholesteatomatous cavity, it seems plausible that these groups of epithelial lined cells might coalesce forming an extension of the original cavity.

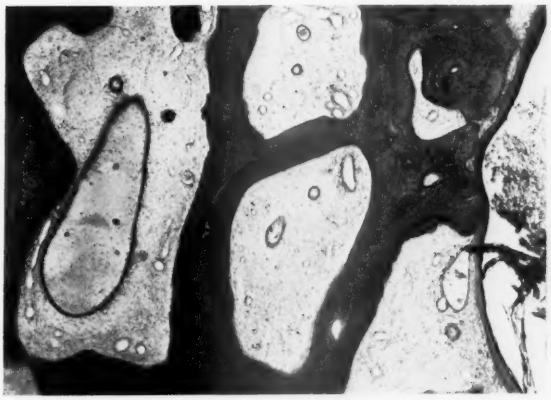


Fig. 6. Case 2.—A detailed picture of the cell group described under Fig. 5, Case 2.

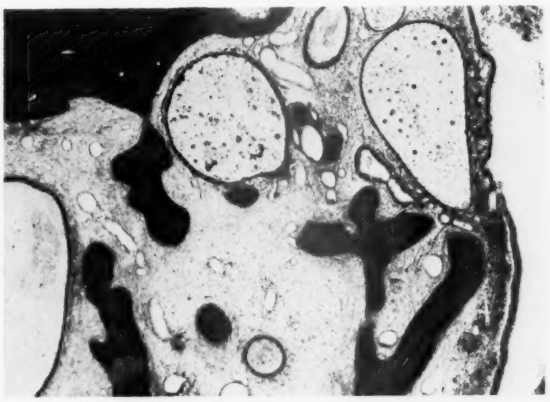


Fig. 7. Case 2.—Peritubal cells in the anterior portion of the perilabyrinthine region showing the same pathology as those cells in Fig. 5 and Fig. 6, which are in the postlabyrinthine or mastoid region.

Fig. 8. Case 3.—Vertical section of the perilabyrinthine region, showing a severe middle ear involvement with pus in the epitympanic and hypotympanic cells. The process diminishes in severity as it progresses around the labyrinth and forward into the petrous pyramid. The subarcuate cells also show pathology which extends forwards. One large pneumatic labyrinthine cell is seen to be filled with pus, and is obviously a continuation of a chain leading to the middle ear cavity. The eminentia arcuata is a cellular formation and gives little information regarding the position of the underlying superior semicircular canal. Procedure based on measurement of the petrous pyramid from this landmark might therefore be erroneous.

Fig. 9. Case 3.—A detailed picture of the junction between the epitympanic and perilabyrinthine cells showing a gradual diminution of the inflammatory exudate and the thickness of the lining membrane as the distance from the original process increases.

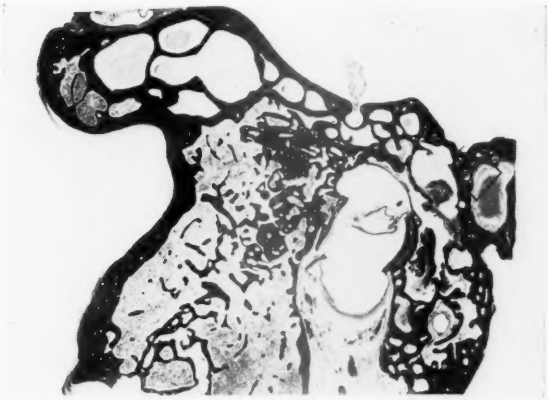


Fig. 10. Case 3.—Vertical section in the antelabyrinthine region showing exudate in the superior petrous cells extending mesially to the petrous ridge. This picture also shows a dural extension into the crevice on the superior surface of the petrous pyramid. It is especially interesting to those who elevate the dura from the floor of the middle fossa in order to gain access to the antelabyrinthine petrous cells. In addition, it is obvious what would happen if the thin, bony layer were destroyed by a progressive infection.

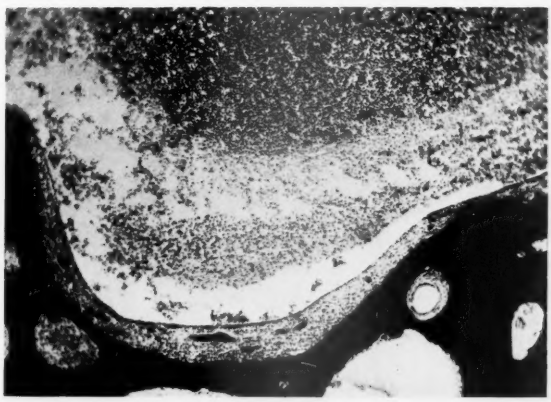


Fig. 11. Case 3.—A detailed picture from Fig. 10, Case 3, showing a peritubal cell filled with pus.

Fig. 12. Case 3.—A vertical section cut at the level of the perpendicular portion of the facial nerve in the postlabyrinthine or mastoid region. This shows a severe purulent destructive involvement with the gradual diminution of the severity beyond the area of active destruction.

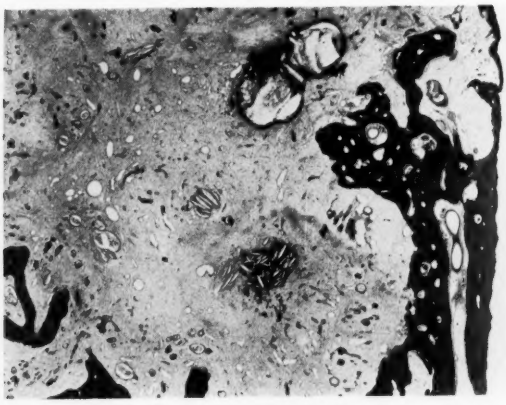


Fig. 13. Case 4.—Vertical section in postlabyrinthine region demonstrating fibrous granulation tissue with organization. Spaces which were probably occupied by cholesterin crystals are present.

Fig. 14. Case 5.—High magnification showing pathology similar to that in Fig. 13. This section is from the antelabyrinthine region of the petrous pyramid.

Fig. 15. Case 6.—A section through the eustachian tube showing infected peritubal cells.

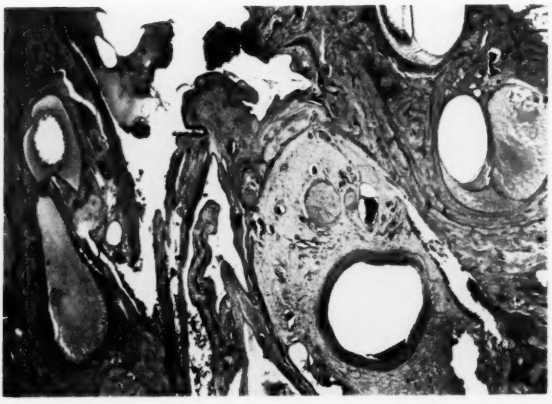


Fig. 16. Case 7.—In this picture the peritubal cells are involved together with the pericarotid area and the labyrinth. Histologically this is an old inflammatory process with an acute exacerbation.

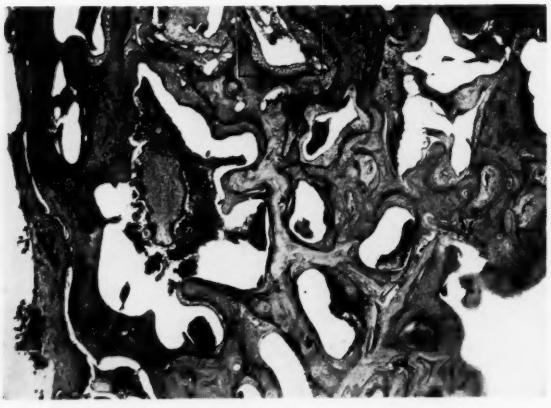


Fig. 17. Case 8.—Horizontal section in the antelabyrinthine area showing thickened membrane, purulent secretion and bone destruction, due to an acute infection.

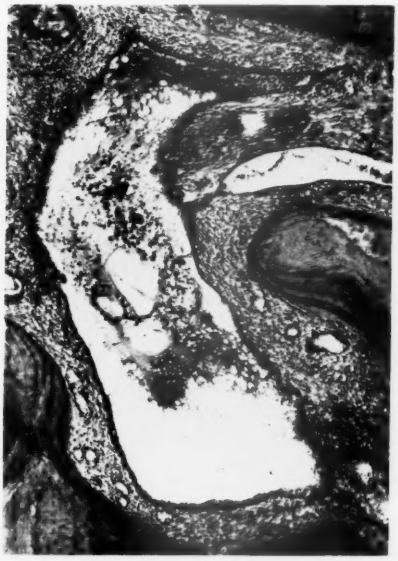


Fig. 18. Case 8.—High power from area marked out in Fig. 17.

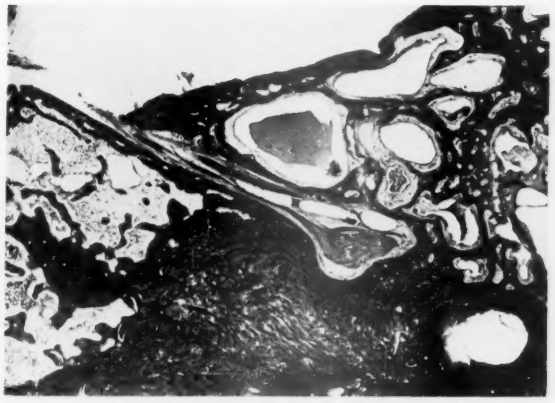


Fig. 19. Case 9.—A section through the geniculate ganglion and the greater superficial petrosal nerve. Pneumatic cells posterior to the nerve give evidence of a prolonged involvement with a superimposed recent process. The cells anterior to the petrosal nerve are marrow containing. It is in cases similar to this one that the malignant type of petrositis develops. The original invasion takes place through a small chain of cells, then gains space in the larger pneumatized or marrow areas. The virility of the infection is diminished and it lies dormant. In the meantime fibrous organization may occur along the small chain of cells through which it gained entrance, and cause an obliteration of the pathway with subsequent infection, but the free discharge of pus is obstructed. The result is the formation of a new avenue of escape for the confined pus.

CVIII.

J. Gordon Wilson, M. D.,

Chicago.

In my discussion I confine myself to the petrous tip, and only refer to the adjacent parts in so far as they cause or modify, directly or indirectly, pathologic processes in that area or have a bearing on anatomic structures. To outline clearly the position of this area, it lies medial to the internal auditory meatus and the bony capsule of the cochlea and terminates at the synchondrosis of the basi-occipital and the sphenoid. Its apex (apex pyramidis) is inserted in the angle between the occipital and the sphenoid, leaving an irregular chink (foramen lacerum) filled with fibrocartilage. The inferior portion constitutes a part of the under surface of the base of the skull and is in close relation to the canal of the internal carotid artery, which canal encloses sympathetic nerves and a plexus of veins communicating freely with the veins of the tympanic cavity.

The following study is based on an examination of the petrous tips of fifty children, aged from five weeks to fifteen years. It is incomplete in so far that the extreme tip had been removed in about 50 per cent, in which the bones had been prepared for other investigations; but in all there was present the area medial to the cochlea and the internal auditory meatus, comprising half of the tip. These ages were chosen for this preliminary report because, in my experience, symptoms of inflammation involving the tip (petrositis) are most common during these ages.

The development of the petrous bone is related to the growth of the cranial cavity; it changes in size and in position with corresponding changes in the skull. As a result, the auditory capsule is slightly displaced towards the base. From measurements we have found its length in the adult is on the average one and one-half times as great as that of the bone in the infant and its width may be regarded as doubled. During development the petrous bone might be considered as the pivotal part of the floor of the skull. From its position it is subject to strains, probably more than the other bones of the skull, the direction of the strains being indicated in general by the direction of the trabeculæ. We have then this interesting picture: while the membranous labyrinth with its primary osseous capsule remains unaltered in size after birth, the petrous tip undergoes marked increase in size, due to bone formation under the periosteum,

and to growth between the otic capsule and the basi-occipital and sphenoid. With this increase there is a corresponding change in the trabeculæ and a widening of the spaces which separate them and contain marrow cells. As elsewhere, the bone marrow in the young is predominantly of the red variety, in the adult yellow (fatty). The amount of red marrow, both in the young and in the adult, may be greatly increased by local irritation associated with disease in neighboring tissues or elsewhere in the body. The petrous marrow has an abundant blood supply, which comes chiefly from the arterial plexus in the carotid canal, but vessels also enter from the dura and from the tympanic cavity.

When one examines that part of the petrous bone which lies medial to the primary osseous capsule of the labyrinth, the character of the bone is noteworthy. A secondary circular deposit of bone is formed, external to the primary osseous capsule but of different histologic character. At first relatively wide marrow spaces separate these two, but with age these intervening spaces diminish in width and almost disappear, though not entirely. This secondary capsule is compact towards the cochlea but opens up medially, giving place to trabeculæ forming the reticular structure (cancellous bone), so obvious in dry preparations. In the young these trabeculæ are close together; with development they become wider apart; in both the lamellæ towards the tip are thinner. In all the marrow spaces communicate freely. In the developing bone, osteoclasts are to be seen.

A varying degree of pneumatization at the tip has been described. The following may be taken as an example: "There have been frequent demonstrations of almost complete pneumatization of the petrous tip. Occasionally large cells are present which practically hollow out the entire apex." (Meltzer, Trans. Am. Otol. Soc., 1930, p. 226.) The question of pneumatization some years ago might have been regarded as of academic interest. Now it is important because the treatment of inflammation and suppuration in pneumatic spaces and of similar lesions in marrow cavities are not regarded by surgeons as fundamentally identical. Infected pneumatic spaces may be curetted and broken down; the curetting of infected osteomyelitic areas, especially in the acute or subacute stages, is not in keeping with present surgical procedure.

In discussing this subject I take it that we are agreed that no diagnosis of a pneumatic space can be made except under the microscope; the spaces in a dry bone offer insuperable difficulties to the settlement of this question. There must be identified the enclosing wall

characteristic of an air cavity wherever found, namely, epithelium. Wittmaack, who first studied their formation, showed that they are formed by invasion of epithelial elements from the middle ear. The formation of pneumatic (air) spaces can be clearly followed in a developing mastoid, where one can see the bony walls of the marrow (diploetic) spaces adjacent to an air cell breaking down and the epithelium from the air cell gradually displacing the marrow to form a pneumatic space. This holds not only for the mastoid but for developing air spaces in other areas in the middle ear, including the region of the eustachian tube. I have not seen such a developmental progression in the osteomyelitic spaces of the petrous tip. Pneumatic cells occurring in the tip must develop from pneumatic mastoid cells not uncommonly seen lying close to the floor of the skull lateral to the internal auditory meatus or from tympanic pneumatic cells; I have seen no evidence that they do so.

Pneumatic cells are frequently seen in children external to the internal auditory meatus, close to and even internal to and above the semicircular canals (Fig. 1). These are mastoid cells which have developed into diploetic spaces at the base of the skull, and ought so to be regarded. They are frequently involved in mastoid suppuration, even when the mastoid tip is little affected. But my examination has not shown that they progress medial to the cochlea or internal auditory meatus. I would have you note the labyrinthine bone which separates mastoid cells from the petrous tip, a solid mass with few and small marrow spaces, forming an effective barrier to the progression of air spaces. In microscopic preparations it occasionally happens that owing to the plane in which the sections have been made, a pneumatic cell at the base of the skull and part of the mastoid series seems to lie medial to the internal auditory meatus, but an examination of adjacent sections shows the exact location of this cell and corrects the first conception. Mere size is no criterion of a pneumatic space; large marrow spaces in the apex are to be found not only during disease processes but also after disease. Occasionally one notes in this region spaces in which no marrow is present and in which the walls are of thin fibrous tissue, but these appear to me to be cysts which have followed inflammatory effusion into marrow spaces. Again, one may see towards the middle ear a shallow space having the appearance of an anatomic air cell but which I believe to be an abscess cavity which has ruptured, healed and been converted into an epithelial lined cavity. I have examined pneumatic cells associated with the eustachian tube, but none of these perforate the bone-confining wall of the apex and displace the marrow cells to constitute a definite anatomic pneumatic space.

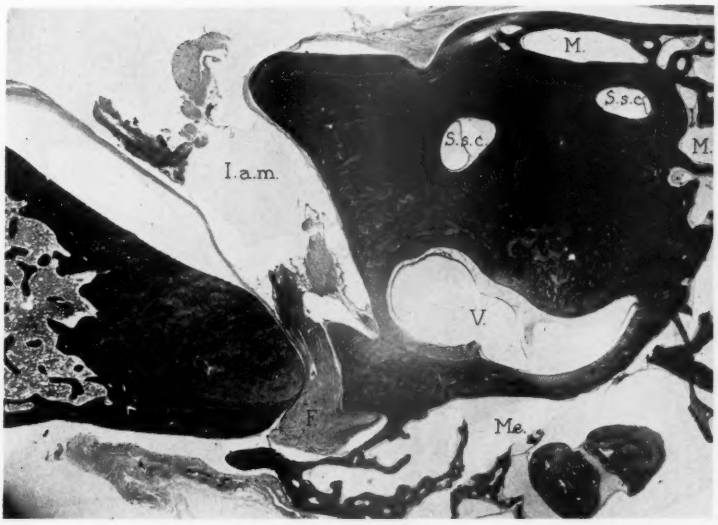


Fig. 1. Child of 3 years (December 22, 1933—slide 190), with normal middle ear and mastoid, showing pneumatic cells at base of skull extending over superior semicircular canal, also pneumatic cells in tympanic cavity. *I. a. m.*, internal auditory meatus; *F.*, VII nerve with geniculate ganglion; *V.*, vestibule; S. *s. c.*, superior semicircular canal; *M.*, mastoid cells; *A.*, petrous apex with marrow; *Me.*, middle ear with the malleus incus joint.

That pneumatic cells do exist in this apical region appears to be assured from the investigations of competent observers. Gradenigo believed that the majority of the cases which presented his syndrome had pneumatic spaces extending into the apex. I have not seen them in the petrous tip of children, varying in age from birth to fifteen years, the age period where symptoms of inflammations at the tip are most common. It would be interesting to know what percentage of pneumatic cells described at the tip in children under fifteen years develop in pathologic bone.

The following brief case report and postmortem findings illustrate some of these points:

REPORT OF A CASE.

CASE 1. J. P., age 13 years. At the age of 18 months had right otitis media with ear discharge for seven weeks. Was apparently well till July, 1934, when he again had a right otitis media with otorrhea for six weeks. On December 7, 1934, the ear

Fig. 2. Child, 13 years, died of meningitis preceded by paralysis of R. VI, and pain in right temporal region. Right petrous bone divided perpendicularly and lateromedially about 6 mm. anterior to superior petrosal sinus, showing cavity and with fibrous tissue and some pus almost filling the tip and connected with cartilage of foramen lacerum. (See above.) *A.*, abscess cavity; *C.*, cochlea; *G.*, gasserian ganglion; *V.*, vestibule; *J.*, jugular sinus; *M.*, part of mastoid antrum.

began again to discharge and this has continued. The patient was admitted to the hospital February 4, 1935. Two days later a right mastoidectomy was performed and granulation tissue and pus were uncovered. On February 19, the child had slight vertigo and tenderness over the right frontal. No signs of meningitis were present. On February 20th, the mastoid was reopened; examination showed no disease in the cranial cavity or zygomatic cells. On February 26, there was a right frontal headache and right sixth nerve paralysis with signs of meningitis. Bronchopneumonia developed and the patient died on March 4, 1935.

Postmortem examination (Fig. 2) showed a large cavity occupying the entire petrous tip, full of fibrous tissue and granulation tissue with some pus. It was enclosed by bone except towards the apex, where there was a small fistulous tract under the Gasserian ganglion lying along the course of a venous channel communicating with the superior petrosal sinus. The floor of the abscess cavity was glistening in appearance towards the apex and connected with the fibrocartilage of the foramen lacerum. The bone between the abscess cavity and the dura was 1 mm. thick; bone, normal in appearance and about 3 mm. in thickness, separated the abscess cavity from the cochlea. There was a clot in the jugular foramen; the carotid canal and artery were normal. The history, together with the postmortem appearance suggests a recrudescence of an old osteomyelitic process which flared up when a secondary infection occurred.

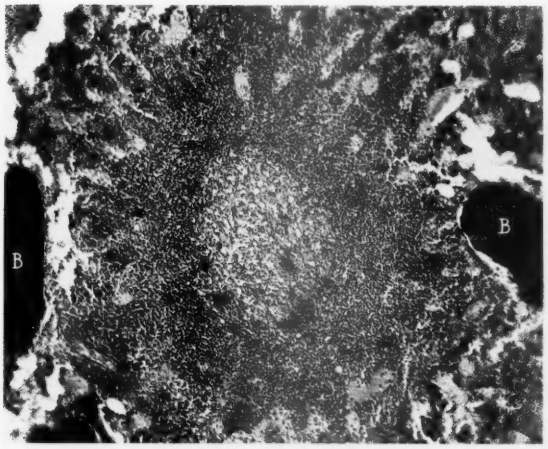


Fig. 3. Child of 5 years (July 30, 1932), Slide 235-1, with normal middle ear and mastoid; died of abscess of lung and pericarditis secondary to osteomyelitis of left tibia. Active hyperplasia in marrow of petrous tip. Core-like formation with radiating columns containing hypertrophic connective tissue, suggesting destruction of tissue. *B.*, bone trabeculæ. Magn. 95 diams.

Petrositis, as otologists see it, is a localized osteomyelitis usually associated with pathologic changes in the middle ear. The cells of the marrow filling up the interstices of the bone are greatly increased in number and serous effusion can be noted. Subsequent to this the calcified framework is involved in the inflammatory process, the bone cells and the calcified tissue showing signs of destruction (necrosis). The early signs are similar to an acute infection elsewhere: engorgement of blood vessels, exudate of serum and emigration of red and white blood corpuscles. Pus and granulation tissue appear and the necrotic bone of the trabeculæ is absorbed, leaving an abscess cavity which may be small or may involve a large part of the tip (Fig. 2). As the subacute or chronic stage subsides, the cavity may persist and become lined by granulation and later fibrous tissue. The pus may remain for an indefinite period, or be replaced by a serous exudate with a gross appearance very similar to that of a small cyst. The exudate may be entirely replaced by granulation tissue, which in turn may mature into fibrous tissue. The periosteal bone, even to a late period, may be intact; if the disease persists it becomes thin and necrotic, till finally there is a fistula with the escape of pus.

The micro-organism may reach the tip in one of two ways, by the blood stream as part of a general infection, or by direct extension

Fig. 2. Child, 13 years, died of meningitis preceded by paralysis of R. VI, and pain in right temporal region. Right petrous bone divided perpendicularly and lateromedially about 6 mm. anterior to superior petrosal sinus, showing cavity and with fibrous tissue and some pus almost filling the tip and connected with cartilage of foramen lacerum. (See above.) *A.*, abscess cavity; *C.*, cochlea; *G.*, gasserian ganglion; *V.*, vestibule; *J.*, jugular sinus; *M.*, part of mastoid antrum.

began again to discharge and this has continued. The patient was admitted to the hospital February 4, 1935. Two days later a right mastoidectomy was performed and granulation tissue and pus were uncovered. On February 19, the child had slight vertigo and tenderness over the right frontal. No signs of meningitis were present. On February 20th, the mastoid was reopened; examination showed no disease in the cranial cavity or zygomatic cells. On February 26, there was a right frontal headache and right sixth nerve paralysis with signs of meningitis. Bronchopneumonia developed and the patient died on March 4, 1935.

Postmortem examination (Fig. 2) showed a large cavity occupying the entire petrous tip, full of fibrous tissue and granulation tissue with some pus. It was enclosed by bone except towards the apex, where there was a small fistulous tract under the Gasserian ganglion lying along the course of a venous channel communicating with the superior petrosal sinus. The floor of the abscess cavity was glistening in appearance towards the apex and connected with the fibrocartilage of the foramen lacerum. The bone between the abscess cavity and the dura was 1 mm. thick; bone, normal in appearance and about 3 mm. in thickness, separated the abscess cavity from the cochlea. There was a clot in the jugular foramen; the carotid canal and artery were normal. The history, together with the postmortem appearance suggests a recrudescence of an old osteomyelitic process which flared up when a secondary infection occurred.

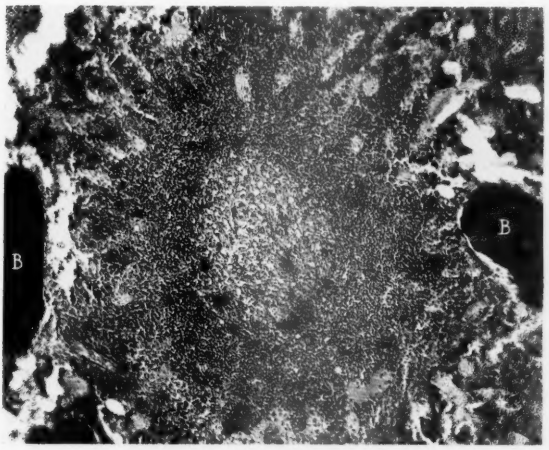


Fig. 3. Child of 5 years (July 30, 1932), Slide 235-1, with normal middle ear and mastoid; died of abscess of lung and pericarditis secondary to osteomyelitis of left tibia. Active hyperplasia in marrow of petrous tip. Core-like formation with radiating columns containing hypertrophic connective tissue, suggesting destruction of tissue. *B.*, bone trabeculæ. Magn. 95 diams.

Petrositis, as otologists see it, is a localized osteomyelitis usually associated with pathologic changes in the middle ear. The cells of the marrow filling up the interstices of the bone are greatly increased in number and serous effusion can be noted. Subsequent to this the calcified framework is involved in the inflammatory process, the bone cells and the calcified tissue showing signs of destruction (necrosis). The early signs are similar to an acute infection elsewhere: engorgement of blood vessels, exudate of serum and emigration of red and white blood corpuscles. Pus and granulation tissue appear and the necrotic bone of the trabeculæ is absorbed, leaving an abscess cavity which may be small or may involve a large part of the tip (Fig. 2). As the subacute or chronic stage subsides, the cavity may persist and become lined by granulation and later fibrous tissue. The pus may remain for an indefinite period, or be replaced by a serous exudate with a gross appearance very similar to that of a small cyst. The exudate may be entirely replaced by granulation tissue, which in turn may mature into fibrous tissue. The periosteal bone, even to a late period, may be intact; if the disease persists it becomes thin and necrotic, till finally there is a fistula with the escape of pus.

The micro-organism may reach the tip in one of two ways, by the blood stream as part of a general infection, or by direct extension

from the adjacent parts. The most frequent demonstrable portal of entry follows a lesion of the mucous membrane of the middle ear. Regarding the path of invasion there is a diversity of opinion. For example: "The infection spreads from the tympanic cavity via a preformed air cell passage or by vascular metastasis or by hematogenous pathway or by retrograde lymphatic current." (Sjoberg, Acta Oto-Lar., 1934, Vol. 19, p. 499.) "It cannot be denied that the majority of petrous pyramid infections which reach the apex do so by direct extension through the pneumatic layer of cells extending along the eustachian tube from the tympanum to the petrous tip." (Friesner and Druss, Trans. Am. Otol. Soc., 1930, p. 223.) After describing four routes by which infection may spread—(1) Sublabyrinthine, (2) through subarcuate fossa, (3) carotid canal, (4) eustachian tube—Perkins says, "Anyone who has made many dissections of the temporal bone will readily see that this list of routes is only partial and that the variety and position of cells is endless." (Perkins, Annals of Otology, Vol. 19, 1910, p. 698.)

From these statements I conclude that regarding the most common routes of invasion one cannot at the present time be dogmatic. The examination of my pathologic specimens shows that the infection has usually spread along vascular channels, and probably lymphatics, from the middle ear. This is based on the following observations: (1) In subacute cases the pathologic reaction is most observable in the part adjoining vascular areas connecting with or adjacent to an infected tympanic space, or infected areas near the carotid canal or eustachian tube, without noticeable bone destruction under the membrane of the middle ear; (2) the internal carotid venous plexus receiving the veins from the middle ear are connected with numerous veins from the tip which may be dilated and in some cases thrombosed; (3) in the postmortem material examined by me the infection could not have extended through pneumatic spaces as these were not present. The lymphatics in this region are not known but probably are connected with the tympanic cavity. A localized osteitis may begin subperiosteally near the middle ear or carotid canal. Then it usually results in the destruction of a small area of cortex, which is absorbed, leaving a shallow depression, at first filled with pus and granulation tissue and later healed with cicatrization.

The disease may be acute, subacute or chronic, often with acute exacerbations. In mastoiditis the infection may not result in a purulent cavity, so in petrositis one may expect to see a nonsuppurative inflammation. If the inflammation has resulted in necrosis, which theoretically may occur in any area, a fistula with the escape of pus

may occur at any point; in the pathologic cases I have examined these have formed usually towards the tip, infra-dural, in two on the antero-inferior surface.

A petrositis may be part of a general osteomyelitis. Fig. 3 shows petrositis associated with a primary osteomyelitis of tibia with middle ear and mastoid normal and no meningitis.

When we come specifically to the causal organism commonly present in petrositis we find no agreement. In five cases, Voss found streptococcus in two, streptococcus and bacterium coli in one, pneumococcus in two. Fowler, in five cases found pneumococcus in three, streptococcus in two. In the majority of reports the organism appears to have been obtained from the middle ear or mastoid. The possibility must not be overlooked that septic organisms, once they have entered the blood stream, may, like the spirochaete and the typhoid bacillus, find their way into the marrow of bone, and lie inert for considerable periods, until some favoring circumstances, such as an inflammation of the middle ear, or a serious depression of health, excited them to activity.

Some years ago I stated that in my opinion the marrow cells in this region have a defensive function equipped to deal with an infection reaching its tissue. This opinion has not been diminished by further observations. I have yet to find a case of otitis media or of basal meningitis in which the marrow cells are not greatly increased, so great in some cases as to completely conceal the delicate marrow spaces with the capillaries. The vessels are engorged and there is effusion indicative of an irritative reaction. It is agreed that myeloid tissue belongs to the reticulo-endothelial system which has not only important metabolic functions but also important defensive functions against micro-organisms. It is known that all general pathologic processes immediately affect the bone marrow; that in most general infections—for instance, during suppuration—the marrow cells are stimulated and the percentage of myelocytes in the bone marrow increases greatly. "There is a growing volume of evidence that in the presence of general body infection the reticulo-endothelial arrangements form one of the most important defensive mechanisms of the body. We find indications of its response in septicemia." (Fraser, Brit. Med. J., Sept. 22, 1934, p. 540.) What factors determine the breakdown of this defense I do not know, but I feel safe in saying that most pathologists agree that a slight or passing infection may well be dealt with without forming a pus cavity. This fact, I believe, should have consideration in our operative procedure.

SUMMARY.

1. The development of the petrous bone is related to the development of the skull and may progress beyond puberty.

2. In this study of petrositis in children pneumatic cells are not present at the tip. Air cells frequently found above the semicircular canals and lateral to the internal auditory meatus are mastoid cells extending along the base of the skull.

3. In my preparations the infection at the tip has most frequently come through vascular channels from the middle ear.

4. Since the marrow is part of the reticulo-endothelial system it is a defensive mechanism and this ought to be considered in the treatment of clinical cases.

CIX.

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NEW YORK.

In the first paper of this symposium it was shown by Dr. Guild that the anatomy of the petrous portion of the temporal bone is quite complicated. Drs. Wilson and Jones have shown that the apical portions of the petrous pyramid are subject to certain pathologic conditions dependent upon this anatomy. Roentgenographic visualization of some of these pathologic conditions has been reported in the literature by Stenvers,[1] Taylor,[2] and others.[3] It is the purpose of this paper to classify and compare the roentgenographic findings in the petrous pyramid by x-ray and the findings in microscopic serial sections of the same or similar bones.

Because of the present usages and misunderstandings it is suggested that inflammation in the apical portions of the bone be called "apicitis" or suppuration of the "surgical apex." This bridges the gap between the surgeons who consider anything anterior and medial to the cochlea as the "apex," or "tip," and the anatomists, who have defined the apex as the most medial and anterior point of the

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*From the Departments of Pathology and Otolaryngology, College of Physicians and Surgeons, Columbia University, with the co-operation of Drs. Golden, Swenson and Abbott of the Department of Radiology of the Presbyterian Hospital. This work was made possible by grants from the Research Council of the American Otological Society and the Hayden-Coakley Fund.

pyramid. In speaking of petrositis (Profant[1]) it is more desirable, as a rule, to avoid the use of the words "petrous apex" altogether and to speak of the petrous pyramid as a whole. "Petrositis" should mean inflammation of the petrous portion of the temporal bone, anterior and medial to the arcuate eminence. It should include the perilabyrinthine as well as the antelabyrinthine cells. To omit the perilabyrinthine cells, as is usually done in speaking of petrositis, is like omitting the mastoid antrum in speaking of mastoiditis; it omits the path of entrance for the disease. If it is desired to be more specific about the locations of suppurations, one can always specify involvement of the infracarotid regions, involvement of the superior angle cells to the anatomic apex, etc. Thus, petrositis becomes a general term[5] like mastoiditis, and important regions of the petrous pyramid are not ignored as possible foci of suppuration.

The variability and inaccessibility of the petrous pyramid make its roentgenography difficult. Unfortunately x-ray plates merely record shadows, and therefore the average roentgenogram of a portion of human anatomy is a series of superimposed shadows. Even with stereoscopic technic it is often difficult in the petrosa to separate one shadow from another and to evaluate the density and outline of each. This is partly due to the fact that most of the petrosal pyramid lies rather deep in the skull and it is almost impossible to obtain a clear picture of its entirety without the superimposition of the shadows of the sinuses, rim of the orbits, or tissues of the neck and calvarium. The main difficulty, however, is shown by serial sections to be inherent in the bone itself. The most common type of petrosa is pneumatized only in a few areas. The surrounding bone is more or less densely trabeculated bone containing marrow. The shadow of this sclerotic or diploic bone, as the case may be, is superimposed on the few pneumatic cells, and if the latter are small or located in certain areas, their shadows may be obliterated completely. Now, whereas in the mastoid, obliteration of pneumatic areas usually means pathology, this obviously does not hold when considering the petrous pyramid. The more highly pneumatized the petrosa the more the problem of estimating the pathology approaches that of the mastoid. If the petrosa is completely pneumatized and an inflammatory process occurs in it, the anatomic diagnosis, as shown by microscopic examination, is "osteitis." This corresponds to the most usual kind of mastoiditis. If the petrosa is not at all pneumatized and an inflammatory process occurs in it, the anatomic diagnosis, as shown by microscopic examination is "osteomyelitis." This type of pathologic process sometimes occurs in the mastoid of children who

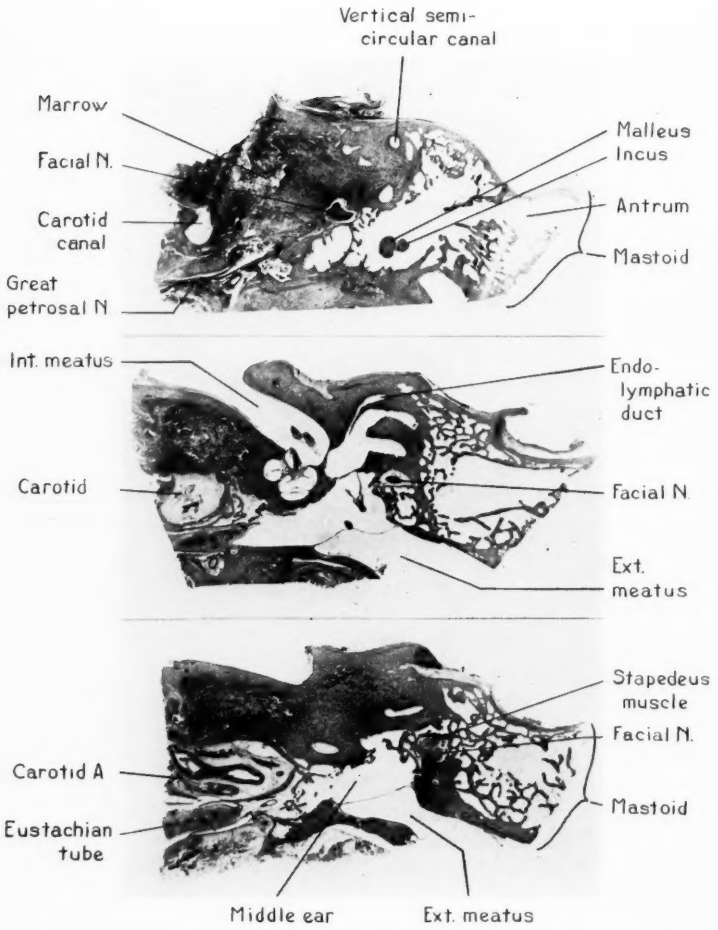

Fig. 1. Case 11217.—Age 17, male, white. Autopsy diagnosis: bacterial endocarditis; no history of ear disease. Horizontal sections through right petrosa, showing hyperplastic marrow in petrosa.

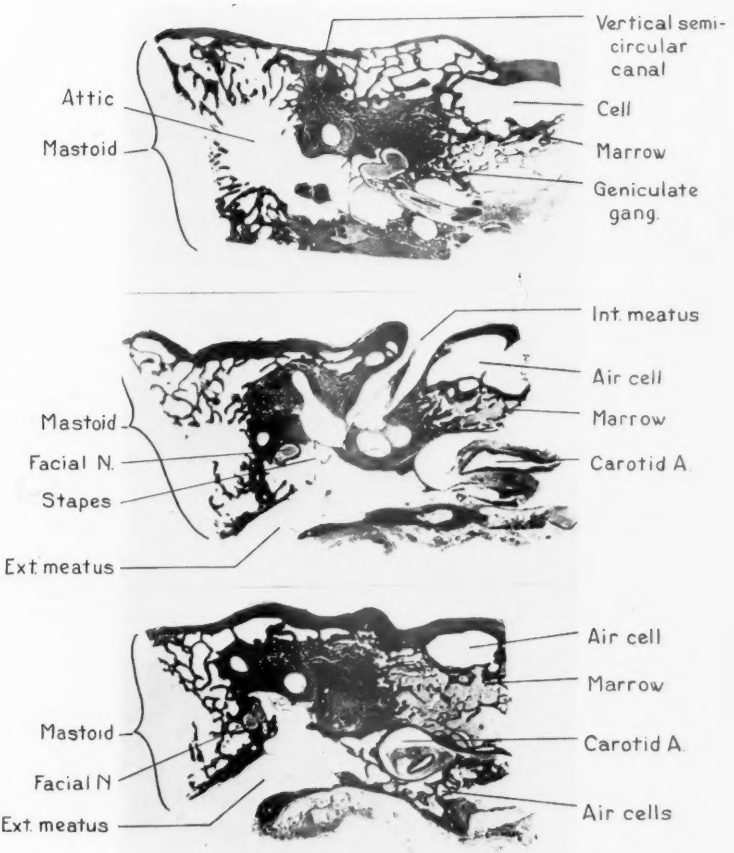

Fig. 2. Case 11259.—Age 34, German, female. Autopsy diagnosis: chronic lymphatic leukemia. Horizontal sections through left temporal bone showing pneumatization medial to superior semicircular canal in (*a*), but less and less pneumatization as the sections go down.

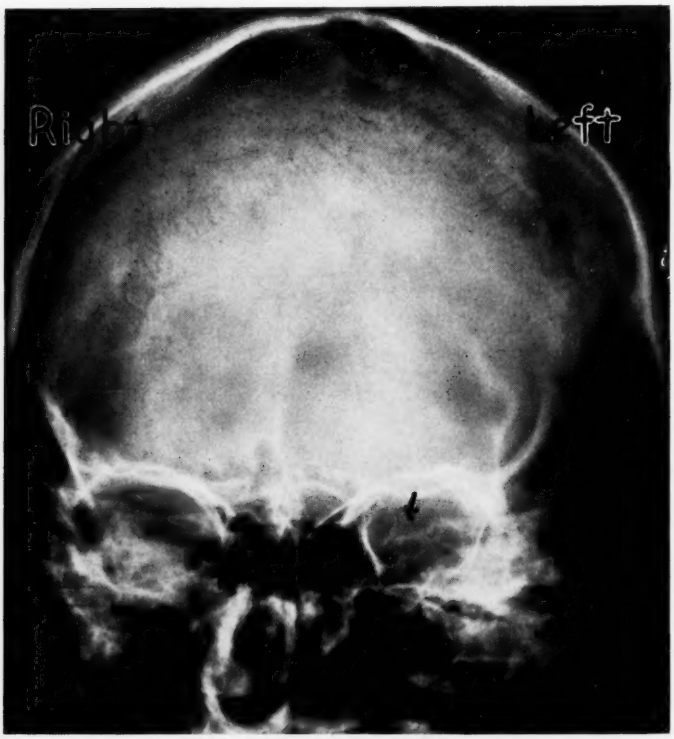

Fig. 3. Posterior-anterior view of case 11259. Note cells in left petrosa projected through the orbit. Compare Fig. 2.

have nonpneumatized mastoid processes, but is more commonly seen as the ordinary osteomyelitis of a vertebra or some other cancellous bone having a relatively thin cortex. The most common type of petrous pyramid, anatomically, is the mixed type, so that in diseased bones we have neither of these conditions in a pure state. That is, in microscopic sections one invariably finds both osteitis of the bones enveloping the pneumatic cells and osteomyelitis of certain parts of the nearby marrow cavities.

Because of this last fact, the first step in the x-ray diagnosis of "petrositis" (i. e., inflammation of the petrous pyramid medial and anterior to the arcuate eminence) is to try to estimate the degree of pneumatization of the diseased side before the disease took place. This can be done in three ways: (1) By comparison with previous,

and therefore presumably more normal x-rays; (2) by comparison with the opposite side; (3) by comparison with the apparent pneumatization of zygomatic and postsigmoid sinus regions. If one or both are highly pneumatized, the petrosa is more likely to contain many perilabyrinthine and antelabyrinthine cells. In addition, highly pneumatized petrosæ are more likely to be wide with an oblique rather than an acute superior angle. In our series of cases these have been found largely in negroes or in individuals of Nordic extraction. There is apparently a higher degree of pneumatization in certain anthropologic types.

It must be emphasized that reliance upon the commonly found similarity in pneumatization in the two sides of the human skull is occasionally dangerous. If there is a history of previous otitis on one side, and especially if this otitis was present in infancy, variations in the pneumatization are to be expected.

Having estimated the probable amount of pneumatization in a suspected bone, one looks for graying of the cell lumen to apparent obliteration of the cell walls by contained fluid or granulation tissue in the early cases (i. e., increased density); for breaking down of the cell walls in advanced cases (i. e., decreased density); for thickening of the cell walls in chronic or recurrent cases; and for breaks in the cortex in fulminating cases. The estimation of the obliteration of the cell contours by contained fluid or granulation tissue is most hazardous if there is superimposed sclerotic or diploic bone. After all, there can be very little difference in the shadows of air cells filled with leucocytes of a purulent exudate, and the trabeculation of diploic bone between which are found various types of hematopoietic cells. If there is pathology in the cancellous bone itself, this becomes apparent in x-ray plates eventually, as does osteomyelitis in a long bone. But in the long bones it is a known fact that it may be ten days or more before an osteomyelitis can be recognized roentgenographically. Only then one begins to find sequestration or a decreased density due to pus pocketing. Later, in chronic cases there may be an increased density due to new bone formation. In the surgical petrous apex all three of these stages occur. Occasionally the labyrinth or part of the labyrinth becomes part of a sequestration.[6] If an osteitis of the pneumatized portion of the bone and an osteomyelitis of the nonpneumatized portion are both present, as is often the case in petrositis, the two conditions cannot be distinguished by any known roentgen technic.

The mento-vertical, or so-called "Taylor position," is at present the most popular position for the study of the surgical petrous apex.

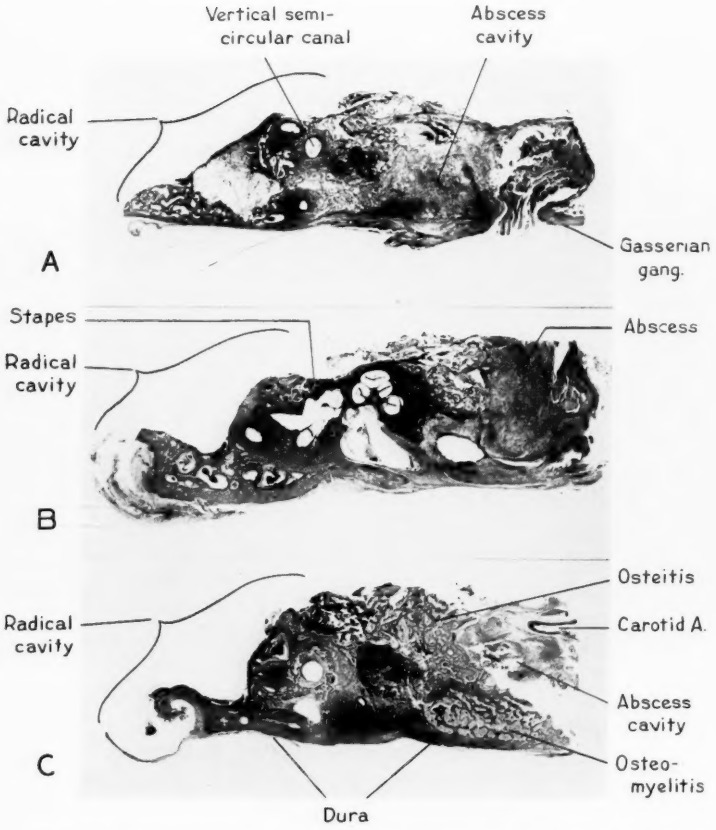

Fig. 4. Case 11522.—Age 41, white, male. Autopsy diagnosis: meningitis of otic origin. Horizontal sections through left petrosal pyramid.

(a) Level of vertical semicircular canal. Radical mastoidectomy cavity partially filled with granulation tissue, fibrin, and leucocytes, and the petrosa medial to the vertical canal filled with similar tissue and necrotic bone.

(b) Level of the vestibule.

(c) Jugular cell region showing inflammatory process extending from mastoid cells to all of the petrosal cells as well as the marrow of the petrosa.

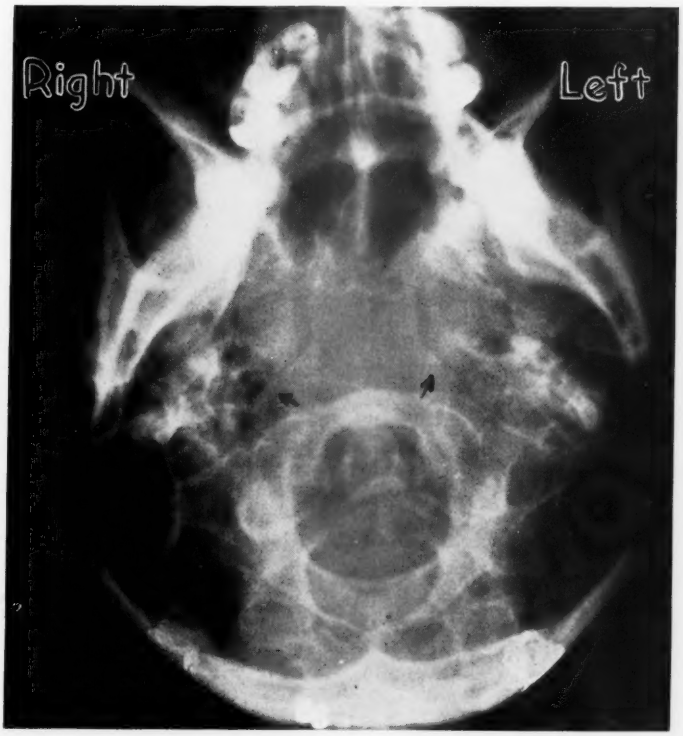

Fig. 5. Case 11522.—Mento-vertical or Taylor position. Note pneumatization on normal right side and decreased aeration on left. Roentgenogram taken after radical mastoidectomy.

It has many advantages, as Taylor[2] has pointed out, the most important being that it allows an over-all view of both petrous pyramids and therefore a comparison between the two sides. The disadvantage is that the projection superimposes the inferior portions of the petrosa upon the superior angle. In our experience most of the cases who die with petrositis have had inflammation in the cells at the superior angle. The fact that some of these cases showed disease in the more inferior portion of the petrosa in addition, does not detract from the importance of this group of cells. Since the superior groups of cells are more likely to be extensive in both normal and pathologic material, it seems that every effort should be made to obtain a clear, uninterrupted view of these cells without the superimposition of other structures which might make it uncertain where the pneumati-

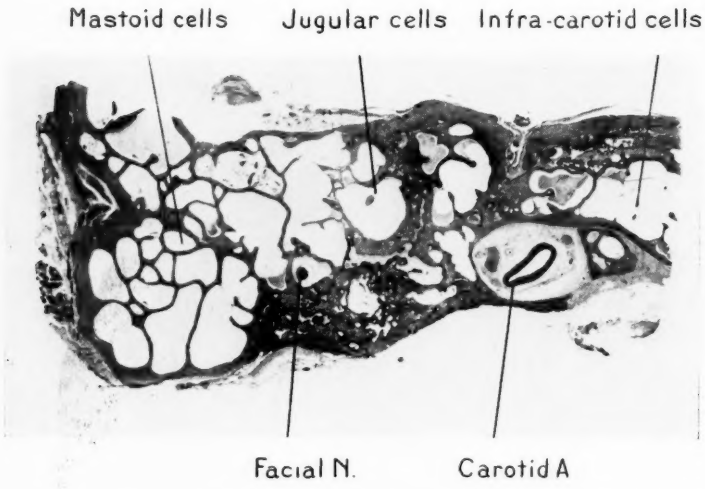

Fig. 6. Case M4.—Age 31, Irish, male. Autopsy diagnosis: meningitis of otic origin. Case of Dr. J. C. Sharp, Manhattan Eye, Ear and Throat Hospital. Left normal side: highly pneumatized petrous pyramid.

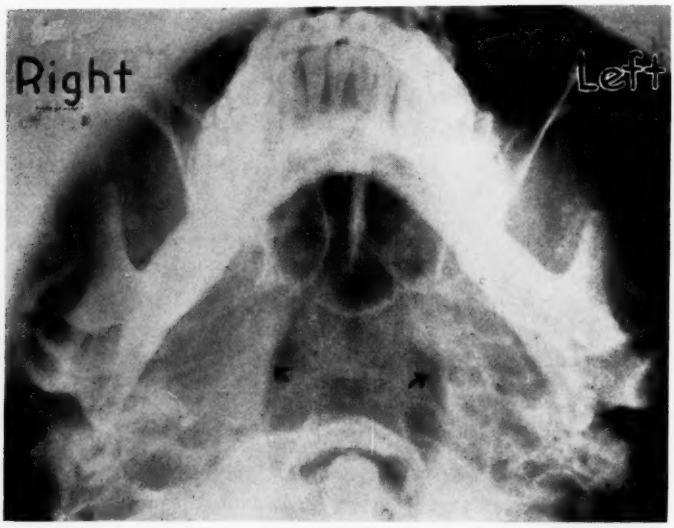

Fig. 7. Case M4.—Mento-vertical position. Courtesy of Dr. F. M. Law. Shows pneumatization on left and decreased density with loss of cell outlines on the right. Compare Fig. 6 and Fig. 8.

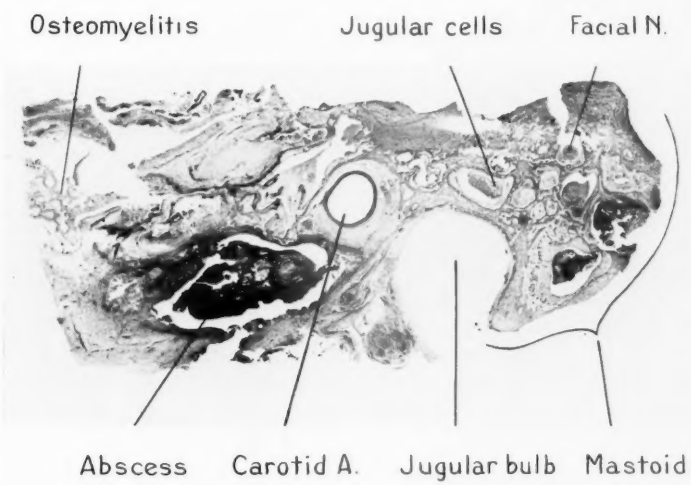

Fig. 8. Case M4. Right side: note large abscess in jugular cell region. Compare Fig. 6 and Fig. 7.

zation lies. A posterior-anterior view, with the superior angle of the petrosa thrown into the middle of the orbit, gives a clear view of these cells and allows a comparison of the two sides. The oblique view, suggested by Stenvers,[1] also gives a good view of the superior angle but necessitates the taking of two plates for comparison of the two sides. Base films which throw the superior angle into the occipital region, also give a good view of the superior group of cells and occasionally help to locate the perforation of the cortex over them. This position, however, attenuates the petrosa in an antero-posterior direction. Because of the complexity and seriousness of the problem it would then seem advisable, if possible, to take stereoscopic projections in the Taylor position for an over-all bird's-eye view and films in the P. A. and oblique projections for further localization. Films for future comparison should be taken as early as possible during the disease, so that a better knowledge of the normal pneumatization of the individual case may be obtained. A record of the exact technical factors used (i. e., milliamperage, kilo voltage, tube distance, exposure time, etc.) should be kept so that subsequent films can be reproduced with the same technic. Serious mistakes can be made by interpreting the changes in the density of films when the technic is not standardized. For example, in case 11,522, a diagnosis of early petrositis was made on the admission x-ray. The patient developed a Gradenigo's syndrome which cleared after a simple mas-

toidectomy except for excruciating unilateral temporal pain. X-rays in the Taylor position, taken three weeks after operation, showed apparent diminution of the process in the petrosa and were so reported. Because of this report and the fact that the ear discharge had greatly diminished, the fever had gone, and the sixth nerve paralysis had cleared, the pain behind the eye and the temple were discounted and the patient was allowed to go home. He returned two weeks later with a meningitis and died soon after admission. Review of the petrosal films showed that in reality there had been no change in the roentgen picture since the original diagnosis of petrositis had been made. Those who read the films failed to appreciate that the second film was somewhat more heavily exposed than the first and that the apparent clearing was due merely to a difference in roentgen technic. This often happens when an inexperienced person attempts to interpret any type of film.

The roentgenogram may be of aid in the diagnosis of labyrinthitis or of labyrinthine irritation. At times definite evidence of pathology in the perilabyrinthine cells can be seen by x-ray. Of course, there is no way of telling that an invasion of the labyrinth is, in a given case, directly from these cells or whether it is through some other route, but marked clouding of these cells, accompanying labyrinthine symptoms, might lead one to do a radical mastoid operation rather than the more usual procedure of doing a simple mastoid operation. We have in our collection a case in which the labyrinthine wall was broken down and the cochlea invaded by infection in the petrous apex. The petrosa in this case showed an osteomyelitis in the region of invasion. There has been one other case of this type in our wards. He had a clinical labyrinthitis and x-rays showed marked clouding of the perilabyrinthine cells. His acute process was cured by a simple mastoidectomy through a wide opening of the zygomatic cells, and the cells over the antrum. Perhaps the so-called "serous labyrinthitis" is at times caused by inflammation in the perilabyrinthine cells.

It must be borne in mind that in every otitis media there is always more or less infection of the mastoid, and if the petrous pyramid is pneumatized there always is more or less infection of the petrosa. We have seen x-rays which showed quite large areas of decreased density in a superior angle group of antelabyrinthine cells. This case had all the classical symptoms of petrositis, but became perfectly well clinically following a simple mastoidectomy. Similar cases have been reported by Coates[7] and others. The important point is that infection may well be apparent in roentgenograms, but x-ray

changes in the petrosa do not necessarily mean operative petrositis any more than x-ray changes in the mastoid necessarily mean operative mastoiditis.

SUMMARY.

1. Petrositis is defined as inflammation of the petrous portion of the temporal bone medial and anterior to the arcuate eminence.

2. If the words "petrous apex" are used in discussing petrositis they should be qualified by stating whether the "anatomic" or "surgical" petrous apex is meant. The surgical petrous apex is apparently those regions medial and anterior to the cochlea and is a relatively inaccurate term.

3. The petrous pyramid is difficult to study by x-ray because of its depth within the skull and because of variations in its own anatomy.

4. Partial pneumatization of the petrosa is the rule in microscopic serial sections, but the cells are usually not apparent in roentgenograms because of superimposed sclerotic or diploic bone.

5. Extensive pneumatization can be seen in roentgenograms, but may be difficult to localize unless a number of projections are made.

6. In our series of nine cases which died with petrositis there have been infected cells along the superior angle, and in some of the cases these were the only pneumatic cells present in the petrosal pyramid.

7. A plea is made for the recording of the technic used for comparison plates so that subsequent changes in density can be more accurately evaluated.

8. X-ray evidence of pathology does not necessarily mean operative petrositis, and apparent absence of pathology in x-ray plates does not necessarily mean there is no petrositis present.

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III. THE CLINICAL PICTURE AND DIAGNOSIS OF THE VARIOUS TYPES OF INFECTION.

CX.

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At this time our knowledge of suppuration of the petrous apex is so far from complete that it is impossible to present an absolutely accurate clinical picture of this condition. Future experience will probably cause us to change our present viewpoint.

Classification. In this paper I am adopting the classification of Eagleton,[1] which is very comprehensive. (See page 1113.)

1. Reactive and reparative osteitis.

2. Non-suppurative congestive cases—symptoms due to venous stasis.

3. Chronic bone sepsis cases (without macroscopic pus).

4. Abscess of the apex.
   a. Without a tract.
   b. Without a tract. (Acute or chronic—Seydell.)

5. Acute septicemia cases, associated with a continuous positive blood culture and meningitis.

Typical Clinical Picture. The following symptoms, signs, and findings may be regarded as being typical of suppuration of the petrous apex:

1. Neuralgia-like pain, especially over the area supplied by the ophthalmic branch of the trigeminal nerve.

2. A discharge from the ear or mastoid wound.
   a. Recurrent.
   b. Continuous.

3. A low grade sepsis.

4. Frequently a paralysis of the sixth cranial nerve.

5. Roentgen ray findings showing destructive changes in the petrous tip.

The clinical picture, unfortunately, is often atypical and death may ensue without the development of symptoms that would lead one to suspect a suppuration of the petrous apex. The symptoms were typical of suppurative apicitis in only 60 per cent of this series. It is essential, therefore, that we discuss all of the variations of the clinical picture. My assignment is on Cases of Suppuration of the Petrous Apex which were published in 1934, and the statistics which I intend to present cover this period and consist of forty-one cases. I am adding an unpublished case on which I had the privilege to operate for Dr. Carter of Wichita, and three unpublished cases of Dr. Gittins of Sioux City, Iowa.

The onset of the clinical manifestations of suppurative apicitis usually becomes manifest during the postoperative period of a simple mastoid operation, performed to eradicate the diseased tissue and drain what was apparently an ordinary acute suppurative mastoiditis. Much more rarely the symptoms appear during a middle ear suppuration which may or may not be complicated by mastoid suppuration. In the cases reviewed by the author, the appearance of the symptoms was on the average twenty-five days following the primary mastoid operation. They developed with the acute otitis media in only two cases.

Pain deep in the eye, or immediately around the eye, is quite characteristic when present. The pain is usually nocturnal in character and agonizing in degree, but with periods of complete absence of pain. In my series there was no history of this type of pain in 36 per cent of cases. Complete absence of pain (hemicranic) was recorded in only 5 per cent. A low grade sepsis was present in 70 per cent.

Many writers have reported cases of suppurative apicitis in which the discharge from the ear or mastoid, or both, subsided, to recur with trigeminal pain, fever, etc. This sequence of events occurred in 41 per cent of this series, whereas in 46 per cent there was continuous discharge. The cases reviewed substantiate the findings of a number of authors relative to the fact that palsy of the sixth nerve is not a definite part of the picture of suppurative apicitis. It was present, however, in about 31 per cent of my cases, usually after

mastoid operation, the longest interval being sixty days. In three cases average time was on the twenty-seventh day following the primary mastoid operation, the longest interval being sixty days. In three cases palsy of the sixth nerve appeared quite early. In one instance, the palsy occurred before the mastoid operation, in another case on the second day, and in the third on the eighth day after the primary mastoid operation. The ages of 89 per cent of the cases with suppurative apicitis fall within the first four decades of life; 34 per cent of the patients were under ten years of age. Apicitis occurs with equal frequency in both sexes. The right and the left ear were involved in practically the same number of cases. Chills were present in 1 per cent and labyrinthine symptoms in 2 per cent of the cases; in the latter group the symptoms were for the most part of a temporary nature. Signs of meningeal irritation or infection were found in 34 per cent of this series.

COMPLICATIONS.

1. *Meningitis.*—The most frequent and important complication of suppurative apicitis is meningitis, which caused twelve of the fourteen deaths in this group. In eleven (Cases Nos. 4, 5, 6, 13, 16, 17, 18, 19, 20, 21, 41) of the fourteen deaths petrositis was not recognized as the cause of the terminal meningitis until the postmortem examination. This fact substantiates Eagleton's statement that petrous apicitis is a frequently unrecognized cause of meningitis.

2. *Facial Paralysis.*—Facial paralysis occurred independently of any operative procedure in one case. It followed radical operations in two cases. In one the technic of Ramadier was employed; in the other, that of Almour.

3. *Deafness.*—In one case sudden deafness and facial cramps occurred, due to an involvement of the nerves in the internal auditory meatus. In one other case deafness, labyrinthine irritation, and facial paralysis followed an operative procedure; the technic of Ramadier was employed.

4. *Brain Abscess.*—In the literature there are a number of statements concerning the rarity of brain abscess complicating infections of the petrous tip. There are three in this series. All were located in the temporal lobe; of these one survived and two died of terminal meningitis.

5. *Impaired Hearing.*—There is but little comment in the literature with reference to the end results as regards hearing in patients who have had a simple mastoid operation and enlargement of an

existing fistula. Kopetzky[2] states: "Let us view these three cases in the light of the advisability of avoiding a radical mastoidectomy; all three have poor, if not to say, bad hearing." (A simple mastoid operation was performed in these three cases.) In two other cases of this series in which the hearing was tested subsequent to operation (Cases 2 and 3), the hearing after convalescence was reported as good. There was no report on the end result in the hearing in Kopetzky's[2] tenth case (Case No. 36), in which he also performed a simple mastoid operation with an enlargement of the fistula. In Case 38 the hearing was reported normal. In Case 37 an unsuccessful attempt was made to find a fistula leading to the tip. Subsequently a radical operation was advised but refused by the patient's family, due to the fact that she was seven months pregnant and suffering with severe glomerular nephritis. In spite of the long continued suppuration, her hearing remained unimpaired. In Case 40 a simple mastoid operation, with an enlargement of the fistula, resulted in recovery without loss of hearing.

6. *Mortality.*—The mortality in this group of cases was about 34 per cent. This is much higher than it should normally be and can be accounted for by the fact that several authors whose cases have been used in compiling these statistics appear to have taken some of their case reports from hospital and postmortem records, apparently with the idea of showing that suppurative apicitis, even ending in death, may run a symptomless course. It is interesting to compare this mortality rate with that of Kopetzky, who has had thirty-two proven cases of suppuration of the petrous tip with five deaths, a rate of 16 per cent.

There can be no question that roentgen-ray films are of great value in the diagnosis of suppuration of the petrous tip. However, it is as yet not possible to determine by a roentgen-ray film whether surgical interference is essential or necessary.

An x-ray of the petrous tip should be made early in the course of mastoid disease. In the great majority of cases this is before symptoms of petrous tip infection are manifest. This x-ray film is valuable first, because it furnishes a key film, and secondly, it gives us definite information relative to the structure of the temporal bone. When symptoms of petrositis appear, repeated films should be made for comparison to determine the progress of destruction or repair.

X-ray findings should be checked by operative or postmortem findings to establish on a firm basis their value in diagnosis. The method chosen in which to take the picture varies with the indi-

vidual, i. e., Taylor prefers a base plate; many European writers favor the Stenver's position. It seems advisable to make films in both positions. Nager states that in the first or early stage of suppuration of the petrous tip there is a general cloudiness. Later the septa are absorbed or become invisible and finally the outlines of the tip are wiped out.

Many authors, Coates,[3] Nager,[4] Sjoberg,[5] and others have reported cases in which there seemed to be a definite area of destruction in the tip, although the patients recovered after a simple mastoid operation. This may indicate that infections within the petrous apex have a much greater tendency to spontaneous cure than has the associated mastoid infection. Eagleton[8] states that this is due to the influence of the red bone marrow.

When routine roentgenograms are made, it will be more generally recognized that asymptomatic infections of the petrous tip occur rather frequently, as in the cases reported by Coates,[3] Sjoberg[5] and others. Sjoberg[5] states that roentgenograms show changes in the tip in nearly all cases which present the Gradenigo triad.

In conclusion, I will quote Taylor,[6] who says: "The roentgenographic examination is an aid, and while it may yield useful information the final diagnosis does not rest on it. Roentgenographic changes in the petrous apex of a destructive nature associated with symptoms and signs of petrositis indicate a suppurative lesion in the petrous apex, with inadequate drainage. There are similar roentgenographic changes in patients who have no symptoms except a persistent otorrhea, but the latter are in no immediate danger from intracranial complications."

CONCLUSIONS.

1. Suppurative petrositis presented in this series a definite or typical picture in but 60 per cent of the series.

2. Death from meningitis ensued in ten cases where the diagnosis was only made at postmortem.

3. The onset of suppurative apicitis usually occurs late, the average time being about a month after the primary mastoid operation.

4. Sixth nerve palsy is not a definite part of the picture of suppurative apicitis.

5. The hearing is not necessarily affected following suppurative apicitis.

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REPORT OF CASES.

CASE 1.—G. M. C. Female, aged 20. Acute otitis, right ear. Profuse discharge and severe pain in the ear and retrobulbar. Pain was nocturnal and persistent in character. Low grade sepsis. X-ray picture showed a well pneumatized mastoid with destructive changes in the mastoid area and petrous tip. Simple mastoid operation. Patient recovered.

CASE 2.—S. D. G. Case A. Female, aged 8. Acute otitis, left ear. Copious discharge. Acute suppurative mastoid. Well pneumatized mastoid. Simple mastoid operation. Fifteen days later severe pain developed behind the right eye and right side of head. There was a low grade sepsis. The pain and fever continued for nineteen days, followed by recovery.

CASE 3.—S. D. G. Case B. Female, aged 6. Acute otitis. Copious discharge. Acute suppurative mastoiditis. Well pneumatized mastoid. Simple mastoid operation. Following the operation, patient developed septic temperature and pain in the left frontal region and in the upper teeth. Six weeks later the pain became worse and localized behind the left eye; was nocturnal in character. The discharge increased. Fifty-eight days after operation sixth nerve palsy developed. Sixty-eight days after operation a mass developed in pharynx and was incised. A large amount of greenish pus drained from mass. Relief and recovery followed.

CASE 4.—E. W. H. Case A. Female, aged 48. A year previous to death left ear began to discharge and later the right ear. No evidence of mastoid infection. Patient died of terminal infectious splenitis, hemolitic anemia and agranulocytosis. On postmortem examination it was found that both petrous bones were evidently involved. There were no symptoms of petrositis.

CASE 5.—E. W. H. Case B. Male, aged 53. Nineteen days before death from hypertension, cardiac hypertrophy and renal arteriosclerosis, etc., patient developed an acute suppurative otitis media with spontaneous perforation of the right drum. Postmortem revealed that there was extensive destruction of the petrous tip. There were no symptoms of petrositis.

CASE 6.—E. W. H. Case C. Male, aged 64. Acute suppurative otitis media and mastoiditis. Simple mastoid. Few days later meningeal symptoms developed with chills and fever on the sixth day following the mastoid operation. Death. Postmortem. Meningitis with extensive destruction of the petrous tip. There was a perforation of the tip on its posterior and superior surfaces. There were no symptoms of petrositis.

CASE 7.—A. A. S. Male, aged 65. Acute otitis media left. Well pneumatized mastoid. Intense pain left ear and temple. On November 16, 1931, simple mastoid operation. Ear dry. After fourteen days pain and discharge reappeared. X-ray showed rarefaction in apex. Septa not visible. December 30, 1931, intense pain left temple and eye. Abducens paresis. January 8, 1932, condition better. January 14, 1932, pain right ear and right eye. January 16, 1932, right mastoid tender. Chill. January 16, 1932, simple mastoid right ear. No infection of mastoid cells. Thrombosis of right sigmoid sinus. The infection from left petrous tip probably involved some of the venous spaces of the base, traveling to and infecting the opposite lateral sinus. Recovery.

CASE 8.—F. R. N. Case A. Female, aged 26. Acute otitis media right. Entered hospital on July 2, 1932, with chills and mastoid tenderness. X-ray revealed slight clouding mastoid cells. Cell walls in tip destroyed. Variation in size of cell in petrous tip. July 7th, sixth nerve palsy and meningeal symptoms. Simple mastoid operation. Mastoid cells empty. Antrum filled with granulations. Could not find paralabyrinthine cells or fistulæ. Attempted to reach tip by following the upper surface of the petrous—failed. Patent died of meningitis. Streptococcus mucosus. Postmortem. Ascess found in tip. Walls of carotid canal, the Gasserian ganglion destroyed, the ganglion being infected. Diagnosis: Otitis media with primary apicitis. Could have been drained by the Almour or Ramadier method.

CASE 9.—F. R. N. Case B. Female, aged 34. Acute otitis media. Paracentesis. Three weeks' treatment. Pain located in depth of cranium increased in severity. Supraorbital nerve sensitive. X-ray. Cells clearing up but cranial surface of tip seemed partially destroyed.

Three weeks later patient suddenly developed high fever, headache and stiff neck. Spinal fluid contained streptococcus mucosus. X-ray. No change. Simple mastoid. Serous fluid in cells. No fistulæ leading to tip. Death. Postmortem. In region of the internal meatus cells filled with pus. Erosion through to posterior fossa. No abscess in tip. Epicrisis. Faulty x-ray findings. Misinterpretation of quiescent period.

CASE 10.—F. R. N. Case C. Male, aged 26. Simple mastoid operation on September 3, 1931, in fourth week of an acute otitis in left ear. X-ray. Cloudy mastoid cells. Tip normal. Well pneumatized. Frontal headache continuous. Sixth nerve palsy on ninth postoperative day. X-ray September 21, 1931. Cranial surface petrous bone destroyed. September 23, 1931, upper border internal auditory canal destroyed. October 1, 1931, sudden development of facial cramps. Nystagmus and deafness. Second operation. Attempt made by method of Streit to reach tip failed. The operator then followed posterior canal wall. Broke into abscess in region of the saccus. Epicrisis. Destruction of tip could be observed through serial x-ray pictures. Symptoms of a lesion of the posterior border are facial cramps, sudden deafness and vertigo.

CASE 11.—F. R. N. Case D. Female, aged 24. November 11, 1933, acute otitis media, right. Severe earache. Paracentesis gave partial relief. X-ray. Well pneumatized to tip. Cells of mastoid cloudy. November 26, 1933, pain in ear worse—temperature rose, secretion increased. Simple mastoid on November 27, 1933. Pus and granulation tissue. Streptococcus hemolyticus. Pain relieved. December 8, 1933, secretion increased. Neuralgiaform pain right side, especially severe at night. X-ray. Cloudy tip, septa not visible. December 21, 1933, more absorption of septa. December 26, 1933, operation, technic of Ramadier. Hazelnut sized cavity found. Complete relief from pain but labyrinth irritation, facial paralysis and deafness resulted.

CASE 12.—Ed R. R. Case A. Male, age not given. Side not given. Three weeks following a simple mastoid operation a profuse discharge appeared from the middle ear. Mastoid dry. Persistent pain in temporal region and eye. The dura was exposed over the antrotympanic region. A copious discharge from the depth of the wound took place at a subsequent dressing followed by prompt relief from pain. Recovery.

CASE 13.—Ed R. R. Case B. Age and sex not given. Nineteen days after left mastoid operation patient developed severe pain in left temple and external canthus of eye. Lacrimation and occular

distress. On the 22nd day the temperature rose to 102. Pain left temple and in and about left eye. On the 40th day paralysis of the external rectus. Eightieth day, facial paralysis. Death on the 120th day. Postmortem. Destructive ostitis of the superior surface of the petrous bone near its apex. Meningitis. Death.

CASE 14.—S. B. Male, aged 39. Mastoid operation right on March 30, 1932, on account of an acute otitis media. Chilly sensations after operation. Eight days later temperature rose to 103.6. Discharge increased. Intense pain right side head and behind orbit. Chill next day. Negative blood. Symptoms subsided. Nineteen days after operation temporal and orbital pain again became severe, diplopia and ataxia developed. No vertigo or nausea. At noon that day pain suddenly ceased. Partial loss function vestibular mechanism. X-ray. Absence tip of petrous. Two days later operation converted into a radical. Tip entered Almour and Kopetzky method. No pus found. Patient recovered.

CASE 15.—C. H. S. Male, aged 12. Simple mastoid right six years before. Complete recovery. March 10, 1933, mastoid cavity infected. Incision, drainage. March 25, 1933, violent headache, vomiting, restlessness. Temperature 103. Drowsy, rigidity of the neck. Kernig positive. Spinal fluid cloudy, increased pressure, 8400 cells; no organisms. Mastoid wound revised, dura exposed. Symptoms persisted. April 1, 1933, paroxysmal pain around right eye. Radical mastoid. No epidural abscess found at tip. April 6, 1933, two-thirds ounce of pus aspirated from right temporosphenoidal lobe. April 15, 1933, right frontal pain increasing. Again investigated tip. Abscess found one and one-half inches deep. Discharged May 24, 1933. Recovered.

CASE 16.—E. P. F. Case A. Male, aged 37. Mild earache, left, for four weeks prior to admission. Evening before developed severe pain left frontal and parietal regions. Temperature 106.5. Became comatose. Symptoms of meningitis. Myringotomy, no pus. Spinal fluid, pneumococci, type III. Died within twenty-four hours. X-ray after death. Tip cells left not distinct. Postmortem. Microscopic. Pneumatic cells filled purulent exudate. Signs of chronic inflammation. Microscopic communications with dura.

CASE 17.—E. P. F. Case B. Male, aged 21. Pain both ears—paracentesis, relief, no drainage. Six days later severe pain left ear. Delirium. Neck stiff. Kernig positive. Spinal fluid 5000 cells, pneumococci, type III. Thin fluid drainage from left ear. X-ray. Mastoid cells cloudy. Simple mastoid. Death. Postmortem. Meningitis base,

no macroscopic portal visible in dura. Microscopic dehiscence above carotid canal through which infection invaded carotid artery.

CASE 18.—E. P. F. Case C. Male, aged 24. Two months before admission otitis media with paracentesis. Three weeks later developed right frontal headache. Meningeal symptoms developed thirty-six hours before admission. Pneumococci type III in mastoid. Simple mastoid operation. Died four days later. Postmortem. Meningitis. Dura over mastoid perforated. More medially in region gasserian ganglion bone soft and crumbly. Microscopically all bone filled with purulent exudate. Labyrinth practically sequestrated. Break in wall of the internal auditory meatus caused the meningitis.

CASE 19.—E. P. F. Case D. Male, aged 45. Chronic otitis media, off and on for twenty years. One day the discharge ceased, followed by severe pain right side of head lateral to eye. Three days later developed meningitis. Spinal fluid 3000 cells. No organisms. Simple mastoid, cholesteatoma. Relief for five days, then headache reappeared. Patient died two weeks after admission. Postmortem. Abscess right temporal lobe. Pus could be seen draining into meninges from the petrous tip.

CASE 20.—E. P. F. Case E. Female, aged 54. Chronic otitis media left following scarlet fever at 12 years of age. Diabetic. Radical mastoid to eradicate discharge. Discharge continued. Operation revised one month later. Five months after operation came in with severe frontal and occipital headache on the left side. Attacks of vertigo and fainting. Stiff neck. Babinski positive. Spinal fluid 800 cells, hemolytic streptococci. Death. Postmortem. There was a break in the dura over the petrous. Purulent labyrinthitis and petrositis.

CASE 21.—E. P. F. Case F. Female, aged 35. Developed otitis following scarlet fever at age of ten. Admitted on account of hypertension and nephritis. Developed otitis both ears, especially right. Paracentesis. Hemolytic streptococci. Simple mastoid. Died a few days later. Symptoms of meningitis. Postmortem. Subdural abscess beneath right temporal lobe whose base lay in a petrous tip abscess which had ruptured through the cortex.

CASE 22.—E. P. F. Case G. Male, aged 7. Admitted December, 1930, with otitis media left. Simple mastoid. Discharge continued. One year later radical operation. Middle ear filled with pus and granulation tissue. December 27, 1931, rise in temperature. February 25, pain left eye. Low grade fever. X-ray. Apparent destruction upper

margin petrous tip. Operated March 9. There was an area of necrosis and a fistula in the roof of the eustachian tube which led into abscess in the tip. Uneventful recovery in three months.

CASE 23.—W. S. G. Female, aged 9. On July 26, 1932, following measles, patient was operated on for mastoiditis on right side. Symptoms subsided. August 17, high temperature. Headache extending behind right eye. Partial facial paralysis and sixth palsy. Discharge from ear increased. Reoperation September 2. Fistula found behind and below semicircular canals. Headache and eye pain disappeared. Some nystagmus, spinal fluid negative. Uneventful recovery.

CASE 24.—E. A. S. Female, aged 6. Acute otitis. Paracentesis December 26, 1931. Mastoid operation January 4, 1932. Pneumatic mastoid. Following operation child complained of left retro-orbital pain. Low grade sepsis. Pain worse at night. Vomited once on January 28, 1932. Temperature rose to 103.4. Neck stiff. Kernig positive. Spinal fluid 2000 cells, no organisms. February 1, 1932, radical mastoid. Explored eustachian tube region, no caries. Trautman's angle explored, found necrotic cells leading under labyrinth. Pus found. Patient's general condition improved. A few days later developed a septic temperature. Reoperation. Septic thrombus found in lateral sinus. Removed. Jugular ligated. Uneventful recovery.

CASE 25.—N. v. S. Female, aged 55. Mastoid operation right following acute otitis on August 23, 1932. Pneumatized mastoid. September 8, 1932, favorable progress. At end of September pain frontal, parietal and orbital. Discharge continued from ear. Some nausea. X-ray. Stenvers position, extensive lesion of the petrous portion, trabeculæ broken down. Absence of upper border. September 24, third operation. Radical mastoid, no fistulæ found. Tubal region examined. Impression that pus came through peritubal cells. General condition improved. December 29, pain returned, very severe, rise in temperature. December 31, fourth operation, method of Striet. Extradural abscess at tip. January 10, 1933, sequestrum removed. Recovery.

CASE 26.—J. M. T. Male, aged 17. Simple mastoid operation September 2, 1933, following an acute otitis media. Patient has been suffering with severe pain occipital and parietal regions. The pain persisted after operation, as did the fever and discharge. October 4, 1933, diplopia. October 20, 1933, facial palsy, right. Temperature and pain absent. Spontaneous nystagmus and past pointing to right.

X-ray. Stenvers position. Resorption of petrous tip. Contours of pyramid wiped out. November 23, second operation. Revision of mastoid. No fistula found. Dura raised from upper surface of petrous. No findings. Radical operation. No fistula in tubal region. Upper surface of petrous resected. Dura raised. Medial to semicircular canal prominence, 5 cm. of pus found. Both sixth and seventh palsy improved. Patient recovered.

CASE 27.—S. J. K. Case A. Female, aged 12. On February 3, 1934, following scarlet fever, right purulent otitis. Vomited. Marked neck rigidity. Projectile vomiting. Spinal fluid cloudy and under pressure 1089 cells. No organisms. X-ray. Extensive pneumatization, base plate shows obliteration cell structure on right, with beginning rarefaction at tip. Vertical nystagmus. Simple mastoid. Little pathology. Radical operation. No fistula, epitympanic, antrum or peribulbar. Fistula found at mouth eustachian tube. Probe led to the petrous tip. Dehiscence in superior surface of petrosa. Lipiodol injected into fistula at tubal orifice; this forced two teaspoonsful of thick pus out between superior and horizontal canals. Eye pain disappeared immediately, other symptoms gradually.

CASE 28.—S. J. K. Case B. Female, aged 2. On June 2, 1934, simple mastoidectomy following an acute left otitis. Pain midfrontal area. Fever ranged as high as 104. X-ray showed a suppuration of left petrous apex. Second operation. Revision, no fistula found. Converted into a radical. A fistula was later found at mouth of eustachian tube. This was curetted and packed. Air forced into fistula caused pus to ooze from area located anterior to vertical semicircular canal. Tract curetted. June 6, 1934, temperature rose to 102.6. Dropped by lysis on June 14, 1934. A slight facial weakness was noted which disappeared in six days.

CASE 29.—S. J. K. Case C. Male, aged 6. Simple mastoid operation on right following an acute otitis which complicated an attack of measles. Coalescent mastoiditis. Extensive pneumatization. Had a postoperative low grade otitic sepsis. Vomited twice. Discharged from hospital April 13, 1935. On April 14, 1935, developed pain in right eyeball. A discharge reappeared from ear. Eye pains, nocturnal in character, increased in severity. Fever continued. Diagnosis of suppuration of the petrous apex made on April 11, 1935. X-ray. Definite bone absorption and halisteresis of right petrous tip. Reoperation April 20, 1935. A fistulous opening was found situated behind the superior semicircular canal leading into an abscess in the tip. Uneventful recovery.

CASE 30.—S. J. K. Case D. Male, aged 19. Simple right mastoid operation on February 24 following an otitis media complicating an upper respiratory infection. About forty days later developed a pain in the right eyeball and side of head. Low grade sepsis. Some vertigo. X-ray. Diminution of aeration in basal portion of right petrous pyramid. Apex showed loss of trabeculation, aeration, and decalcification. Profuse otorrhea. Horizontal nystagmus to left. April 23, reoperation. Large fistula found in the quadrant between horizontal and superior semicircular canal. It extended for two and one-half inches into the tip. Low grade septic temperature continued. April 30, external rectus palsy. Swelling lateral pharyngeal wall. Hypoglossal palsy. Swelling was incised and drained. Pus. Convalescence uneventful.

CASE 31.—S. J. K. Case E. Female, aged 15. Right simple mastoid on April 10, 1933, following an otitis media of twenty-one days' duration. X-ray. No evidence of pathology in petrous apex. Extensively pneumatized mastoid. All pain disappeared April 21. On April 23, definite supraorbital pain developed. On dressing mastoid a fistula was found leading inward toward the petrosa and located in vicinity of antrum. Pus was found when the tract was probed. X-ray. Definite osteoporosis with loss of cellular detail. April 25, headache severe. April 26, reoperation. Fistula enlarged. Probe entered one and one-half inches into petrous apex. Supra-orbital pain continued until May 4. Low grade temperature continued to May 12. Uneventful recovery.

CASE 32.—S. J. K. Case F. Male, aged 10. Left simple mastoid on March 8, 1934, following an otitis media which developed after pneumonia and scarlet fever. On March 27 sudden appearance of nausea and right otalgia. Profuse discharge. Severe headache in temporoparietal region. April 4, 1934, mastoid reopened. Headache relieved following revision. Pain returned, especially in and about the right eye. Low grade fever. Suggestive weakness right external rectus. X-ray. Marked decalcification of the right petrous apex with loss of trabeculations. April 11, 1934, reoperation. No fistula found. Radical. Fistula found at mouth of eustachian tube. Lipiodol injected into tract showed a perforation of petrous apex at the inner wall near or above inferior petrosal sinus. Child discharged from hospital April 26, 1934. On May 20, 1934, complained of severe sore throat and headache. May 24, nucal rigidity. Spinal fluid cloudy, 12,000 cells. Culture, streptococcus hemolyticus. Meningitis. Reoperation. Large abscess found in right temporosphenoidal lobe. May 26, par-

tial paresis right external rectus. Bilateral Kernig. Child died June 5, 1934.

CASE 33.—S. J. K. Case G. Male, aged 10. Left simple mastoid in latter part of March, 1933, following a malignant form of scarlet fever. Stormy postoperative period. For four months both middle ear and mastoid drained profusely. Headaches on right side. July 6, 1933, secondary operation. No benefit. Pain in region of left eye. General condition poor. X-ray. Left tip partially decalcified. September 2, 1933, diagnosis empyema left petrous apex. Operation. No fistula found. Radical. Fistula at tubotympanic orifice leading into tip. Probe entered abscess. Uneventful recovery.

CASE 34.—S. J. K. Case H. Male, aged 8. Left simple mastoid on May 26, 1933, following an acute otitis. X-ray prior to operation showed slight degree of osteoporosis. Left tip, no destruction, not well pneumatized. Postoperative low grade sepsis. June 10, pain in left eye. Continued profuse otorrhea. Slight paresis left external rectus. June 15, x-ray. Further decalcification. June 20, reoperation. Radical. No fistula found. Almour operation. Pus found. Impaired function of facial after operation. June 22, abducens palsy complete. Some eye pain. Uneventful recovery.

CASE 35.—S. J. K. Case I. Male, aged 28. On September 26, 1933, a radical mastoid operation right side for chronic mastoid suppuration and pain in right side of head and temporal region. Wound healed. Pain in right temporal region continued. Low grade fever persisted. October 16, pain still present. November 3, pain right temporal region constant. Fever. November 14, x-ray. Suppurative lesion petrous apex. Reoperation November 15. Fistula in region of tubal orifice. Probe entered abscess. Uneventful recovery.

CASE 36.—S. J. K. Case J. Female, aged 10. On December 22, 1933, a right simple mastoid operation following a severe otitis with symptoms of meningeal irritation. Coalescent mastoiditis. X-ray. Aeration right petrous markedly diminished. No evidence of bone destruction. Low grade postoperative sepsis. Middle ear dry, mastoid continued to drain. X-ray, March 26, 1934. Marked diminution aeration of petrous tip, trabeculæ visible. Contours normal. Diagnosis: Suppurative petrositis with adequate drainage. March 29, 1934, reoperation, a fistulous tract found leading into petrosa beneath the posterior semicircular canal. Fistula enlarged, uneventful recovery.

CASE 37.—E. M. S. Female, aged 34. A patient who had been pregnant for seven months and who had suffered with a glomerular nephritis developed an acute otitis media in the left ear late in De-

cember, 1932. A simple mastoid operation was performed on January 22, 1933. The following day she developed pain in the left eye and ear. The pain was paroxysmal in character, excruciating in degree and worse at night. Her temperature was normal. She vomited repeatedly. I first saw her on February 4, 1932. An x-ray taken at this time showed a destruction of the petrous tip. There was a profuse discharge from the external auditory canal. Operation February 5, 1933. Mastoid wound revised. The zygomatic area and the region surrounding the semicircular canals were explored without finding a fistula. No paralabyrinthian cells were found. The dura over the petrous tip was raised and an unsuccessful attempt made to penetrate the tip with a sharp probe. Following the operation the pain continued unabated. The temperature was normal. On February 6, 1932, the patient developed an external rectus palsy. A radical mastoid operation, to be combined with an Almour-Kopetzky operation, was advised but refused by the patient's relatives. The patient's general and local condition gradually improved. She was discharged from the hospital on March 17, 1932. The ear continued to drain for several weeks. All symptoms gradually subsided. Her hearing was but slightly affected.

CASE 38.—T. R. G. Case A. Female, aged 9. Patient had been suffering with pain in and behind the right eye and a discharge in right ear. Simple mastoid operation June 1, 1934. Sixth nerve palsy with double vision two days after the mastoid operation. Low grade sepsis. External canal dry one week after operation. Basal x-ray showed large area of necrosis involving the entire area of the tip. The a. p. view showed that the line of superior ridge of petrous bone was broken. June 23, operative wound revised. Just above horizontal semicircular canal a fistulous tract was found leading to a large cavity. Drained with rubber tissue. June 29, spasmodic pain had disappeared. July 14, no double vision. Hearing is normal in right ear for all forks and whispered voice.

CASE 39.—T. R. G. Case B. Male, aged 31. Patient had otitis media in left ear for eight weeks. Low grade pain in ear and temporal region. Intermittent discharge. April 11, 1932, x-ray showed cells in left mastoid area blurred with some breaking down of septa. Refused operation. April 22, 1932, sudden attack of vertigo, vomiting and spontaneous nystagmus. Patient had fallen. Hearing much reduced in left ear. Basal plates showed some clouding in left tip. April 23, 1932, simple mastoid operation. Pus under pressure. Necrotic bone. Just posterior to horizontal canal sinus was found from which pus came out under pressure when probed. Tract was enlarged

and passed posterior to the labyrinth into a large cavity in the tip. Rubber drainage used. April 24, rotary nystagmus persisted, also headache. Between May 11 and May 15 patient presented symptoms suggesting brain abscess. These subsided spontaneously. Discharged from hospital May 22. Hearing in left ear considerably impaired.

CASE 40.—T. R. G. Case C. Female, aged 10. Following a cold on January 3, 1933, child developed otitis media in the left ear. Spontaneous perforation. Pain became more severe. Simple mastoid operation on January 15, 1933. Continued to have a low grade fever. On January 30, 1933, began to complain of pain in left eye and also stiffness of neck. At this time external canal which had become dry began to discharge. The mastoid drainage increased. Pain and restlessness worse at night. On February 10, 1933, the operated wound was revised. Necrotic bone was found posterior to the horizontal canal. Above the anterior vertical canal a fistula was found and a probe passed through this opening dropped into a large cavity. Wound was drained with a rubber drain. The spasmodic pain and stiffness in the neck did not reappear. Recovery was uneventful. Two years later her hearing in this ear was practically normal.

CASE 41.—H. B., aged 16, sex not given. Child developed an influenza otitis, one week later developed measles. Two weeks following this developed orbital pain and continued temperature. Simple mastoid operation. Large pneumatic cells. One week after operation, orbital pain, headache and fever occurred. Reoperated, zygomatic cells cleaned out. Meningitis, death. Postmortem. Petrositis, subdural abscess and meningitis.

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CXI.

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In an attempt to clarify the clinical picture of suppuration of the petrous pyramid we have reviewed and classified the symptoms and findings of 112 cases reported in the literature during the past ten years. This data together with personal experience with four cases constitutes the groundwork of this paper.

It must be mentioned that these cases have been reported for various reasons, some to emphasize a particular symptom or group of symptoms, others to differentiate or distinguish this clinical entity from the oft-confused Gradenigo's syndrome. Some of the case reports do not contain important data and doubt may be entertained as to the accuracy of the diagnosis in many. Of the 112 cases cited 71 per cent recovered, and 29 per cent succumbed as a result of complications.

The onset of the eye pain following mastoidectomy averaged 17.3 days in the 82 case reports mentioning the fact. The pain was emphasized in a majority of reports as being the most constant and characteristic symptom. It is located in and around the eye, in the

temporoparietal region, sometimes reflected to the occiput of the affected side. At first it is usually nocturnal, described as a feeling of deep pressure from behind the eyeball. During the progress of the disease the periods of severe pain may change. It may occur during the day, the severe pain usually lasting a few hours at a time with intermittent periods of partial or complete relief. If questioned carefully, the patient is found rarely to be entirely free from pain in the parietal region. If seen the day following a nocturnal attack it is hard to realize the extent of the suffering described by the patient.

Both Kopetzky[1] and Eagleton[2] stress this peculiar eye pain as being highly significant in making a diagnosis of petrous pyramid suppuration.

Vail,[3] speaking of greater superficial petrosal nerve neuralgia, says: "This neuralgia is of extreme diagnostic importance when we are dealing with a case of suppurative otitis, because one should look upon the complaint of pain around the eye, back of the eye, in the temporal region and back of the head as pathognomonic of irritation somewhere in the course of the greater superficial petrosal nerve and that place can only be in the petrous apex."

In an acute encapsulated type the eye and associated pains increase in duration and intensity until drainage is afforded, by surgical means or by rupture of the cortex of the petrous pyramid allowing escape of the pus into the cranial cavity. In the latter event, the patient usually goes into the so-called stage of quiescence. The stage of quiescence is characterized by a diminution of the symptoms. The patient is relieved from the intense pain, is less apprehensive and feels generally improved. This stage lasts from a few days to two or three weeks, when the terminal stage of acute septic meningitis or epidural abscess follows, depending upon the rapidity with which the discharge invades the cranial cavity.

Besides being the most characteristic symptom, the pain to some extent signifies the type of lesion: should the attacks of pain increase in intensity and in duration, and the intermittent periods of relief become shorter, especially with a lessening amount of discharge, it is evident that one is dealing with the encapsulated type and that surgical drainage must be considered. On the other hand, should there be lengthening intermittent periods of relief, with corresponding periods of normal temperature and a lessening of the severity and duration of the pains, the outlook is more favorable for a spontaneous cure.

Kopetzky[4] points out two types of acute suppuration:

(a) The ones in which the petrositis is encapsulated and acute and constitutes a threat to meningeal infection.

(b) Those in which the infection has broken outward and formed a fistulous tract toward the middle ear.

The latter type of case will heal spontaneously or continue as a chronic suppuration.

Both of these types occur in pneumatized bones.

Eagleton[5] discusses an osteomyelitis extending through marrow-filled cancellous tissue to the apex with abscess formation.

The discharge from the ear is continuous from the onset of the otitis or reappears in from two to six weeks following mastoidectomy. In fifty-three cases collected from the literature where the facts were given it was continuous in thirty-six and reappeared in seventeen. In four cases of my own, two were acute with continuous discharge, and two were chronic, with intermittent discharge over a period of years.

Many patients with continuous aural discharge following mastoidectomy, pain in the parietal region and a low grade temperature, are reoperated with the hope of finding an overlooked focus in the mastoid, an epidural abscess or a perisinus abscess; eventually it is discovered that the discharge has its origin in the petrous pyramid and is finding its way to the middle ear through a fistulous opening.

An overgrowth of unhealthy, poorly nourished granulation in the mastoid or middle ear, found at reoperation, may designate the presence of a fistulous tract leading into the petrous pyramid. These cases require most careful watching with frequent dressings. The amount of discharge taken in relation to the severity of the attacks of pain often indicates the progress of the condition.

The bacteriology in the reported cases was predominantly streptococcus, of the hemolytic variety. Pneumococcus, types three and four, and hemolytic staphylococcus albus followed in order.

The temperature is of a low grade, septic type, rarely exceeding 102° F., and usually the average is 100° F. or below during the day, except in the terminal stage. Normal temperature for two or three successive days is not unusual in the less severe types.

The blood picture of uncomplicated cases shows only that a mild infection is present, causing an increase of leucocytes from 10,000 to 15,000. The eyegrounds usually show no change.

Dependence should not be placed on a single roentgenogram of the petrous pyramid. In cases of acute mastoiditis, occurring in extensively pneumatized temporal bones, the petrous pyramid will usually show a density corresponding to that of the mastoid.

Taylor[6] states that "every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion of the petrous apex."

Law[7] says "that it is not possible to make a diagnosis of petrous pyramid suppuration on the x-ray alone."

A recent case of operative mastoid on the right side showed an increased density of the left petrous pyramid when roentgenographed.

Coates, Ersner and Myers[8] had a similar experience in nine cases showing roentgenographic changes in the petrous pyramid. Case No. 8 of this series with bilateral mastoidectomy showed symptoms (Gradenigo's syndrome) on the left side, while the roentgenogram revealed changes in the petrous pyramid of the right.

As recommended by many otologists, x-ray plates should be made of the petrous pyramid at the time of the first mastoid x-ray, so that they may be available for future use should involvement of the petrous pyramid be suspected. The comparison of a series of roentgenograms of the petrous pyramid in suspected cases, beginning with the original mastoid exposure, and taken at intervals of a few days, is a valuable aid in arriving at a diagnosis.

Nervous irritability is an important symptom. It differs from the early excitable irritability of meningitis in that it is more of a depressing type, characterized by crying and persistent pleading for narcotics to relieve the attacks of pain. The degree of nervous irritability is in proportion to the severity of the symptoms.

Many of these cases have symptoms of labyrinthitis, such as nystagmus, nausea and vomiting. They are usually transitory, lasting only one or two days, and may be easily overlooked. They are caused by the infection passing through the cells overlying the labyrinth.

Facial weakness occurs in some cases; it is caused by an irritation of the facial nerve somewhere in its course through the petrous bone where it comes in close relation with an infected tract of cells. Like the labyrinthine symptoms, it is transitory, mild in character and may pass unnoticed. When present, these are significant symptoms, considered in conjunction with pain, discharge and low grade septic temperature.

Paralysis of external rectus muscle occurred in more than 50 per cent of the case histories in the literature. It is not necessarily a diagnostic symptom, occurring as it does in the Gradenigo's triad, and at times associated with lateral sinus thrombosis. In two of my four cases, sixth nerve paralysis occurred, but only after a prolonged period of pain, low grade temperature and persistent discharge. It is usually a late symptom occurring when the infection has reached the very tip cells of the petrous pyramid, involving the dura over Dorello's canal.

Acute cases of suppuration of the petrous pyramid occur most frequently in association with pneumatized mastoid bones. This fact should be borne in mind at the time of the mastoid operation.

Carmack[9] states: "There is every anatomic reason for extension of an inflammatory process into the cells of the pyramid, with otitis media, similar to that occurring in a mastoid process."

REPORT OF CASES.

Presenting more or less a typical clinical picture of acute suppuration of the petrous pyramid relieved by the removal of obstructing granulation tissue:

CASE 1*.—Mrs. M., 26 years old, white, was operated upon for acute coalescent mastoiditis in the Bryn Mawr Hospital, February 7, 1934. The bone was pneumatic, the cells extending well into the zygoma and deep over the anterior wall of the tip.

Except for parietal pains, which were attributed to an accompanying acute sinusitis, and an occasional rise of temperature to 100°, the course for two weeks following operation was fairly normal. There was still some purulent discharge from the canal and mastoid wound.

During the night of the fourteenth postoperative day severe pains in back of the eye and temple kept the patient awake in spite of large doses of morphin. The next day more pus was present in the canal and mastoid wound. The patient cried a great deal, complained of eye pain and pains in the back of the head. Roentgenogram of the mastoid showed "a few cells remaining in lower anterior portion of the mastoid and in the zygoma. No evidence of epidural abscess." Two days later mild nystagmus was noticed. It could only be seen when the eyes were turned to the extreme position. The nystagmus and some nausea lasted for three days. Examination by a neurologist was negative. Eyegrounds were negative. Two days later, February 25, the neurologist and ophthalmologist re-examined the patient, with negative findings. Blood culture was reported negative.

From February 25 to March 21, the symptoms were much less severe; the temperature was normal for periods of three to six days; the discharge continued from the wound and the canal.

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*This case has previously been reported.

March 21, five weeks after the original operation, the mastoid wound was reopened. The antral region was filled with unhealthy granulation tissue; no infected cells were found. Large areas of the dura were exposed over the temporosphenoidal lobe, and over the lateral sinus but were found normal. The findings were disappointing.

For two or three days following, the discharge and the severity of the pains were lessened. On the third day following the second operation paralysis of the right external rectus muscle appeared, the discharge was more profuse and the pains were increasing in intensity and becoming more continuous. A roentgenogram of the base of the skull revealed definite cellular development in the petrous pyramid of the left side. On the right, the affected side, the detail was obscured, the cell walls being barely visible. A diagnosis of suppuration of the petrous pyramid was made.

For ten days following there was a decided diminution of the severity of the pain. The temperature was normal for periods of three days at a time. The discharge was more profuse, and the patient showed less nervous irritability.

On the 9th of April, twenty days after the second operation, the patient had a very bad day with continuous severe pains and increased nervous irritability. Dr. F. C. Grant, in consultation, found no localizing evidence of brain disease. Dr. Samuel J. Kopetzky, called in consultation, verified the diagnosis of suppuration of the petrous pyramid and advised operation.

The old wound was reopened and converted into a radical mastoid cavity; the middle ear cavity contained unhealthy granulation tissue. Search was made for fistulæ leading into the petrous pyramid. Three were found: First, from the pretubal area with an opening 2 mm. in diameter and extending for 3 cm. into the petrous pyramid; second, from the prebulbar cells extending for 4.5 cm. inward; and third, from behind and above the superior semicircular canal extending into the petrous pyramid for 2 cm. These fistulæ were gently probed. No further operation was thought necessary for drainage.

Immediately following the operation iodized oil was injected into the pretubal fistula and roentgenogram showed it to reach the petrous apex.

The pain gradually lessened until complete disappearance in one week. The sixth nerve paralysis cleared up in two weeks. The discharge from the canal continued in a decreasing amount until October 1, 1934, six months after operation.

Bacteriologic examinations revealed hemolytic streptococcus as the infecting organism throughout the course of the disease.

Presenting a fairly typical clinical cycle of acute encapsulated suppuration of the petrous pyramid:

CASE 2*.—Male, colored, 46 years old, was operated upon at the Bryn Mawr Hospital for an acute mastoiditis of the right side. The two weeks' postoperative course was normal except for a continuation of a slight purulent discharge from the ear canal. At the end of two weeks he complained of pain in the right temporoparietal region, with an occasional pain around the eye of the corresponding side.

The pain continued, with an increasing intensity at night; there was a slight rise of temperature, and continuation of the discharge from the canal. About ten

*NOTE.—This case is not complete in detail, the hospital record being mislaid.

days following the onset of the pain, an external rectus muscle paralysis of the right side appeared. The symptoms continued for about one week after the onset of the paralysis of the sixth nerve, when suddenly one night the patient was relieved of his pain. When seen on the following day, he and those caring for him expressed the belief he was much improved.

Three days later there was a sudden elevation of temperature, delirium coming on shortly thereafter, and the patient died from a septic meningitis.

This case presents the clinical cycle of acute suppuration of the petrous pyramid with a short quiescent period.

CONCLUSIONS.

In the clinical picture of acute suppuration of the petrous pyramid we find: Attacks of pain, in back of, and around the eye, and in the temporoparietal region, associated with:

1. Continuous or reappearing aural discharge.

2. Continuous low grade septic temperature.

3. Nervous irritability of the patient.

4. Increasing involvement of the petrous pyramid by the infection as shown by a series of roentgenograms.

5. Frequently—External rectus muscle paralysis, transitory nystagmus, nausea and vomiting and facial weakness are found.

CXII.

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Chronic infection within the petrous pyramid presents the pathologic picture of a subacute suppuration. Clinically, it is an acute process that has quieted down and become symptomless except for a persistent otorrhea.

When one realizes that an encapsulated suppuration within the apex can produce a definite set of acute symptoms, and that spontaneous rupture of this encapsulation through a large fistulous tract can give permanent symptomatic relief, then we have the underlying picture of a beginning chronic petrous infection, and the term chronicity applies as long as the acute symptoms are absent and the otorrhea persists.

The clinical picture of a chronic petrous infection is as follows: There has been a preceding acute tympanomastoiditis in a well-pneu-

matized mastoid and petrous apex. After a simple or even a radical mastoidectomy, the symptoms of an acute infection in the petrous pyramid (namely, first division pain, headache, profuse otorrhea, particularly through the external auditory canal, fever, and a possible external rectus paralysis) may disappear, leaving only a persistent otorrhea, which can be attributed to no other cause than a lesion in the petrous bone. The acute symptoms often subside without any operative procedure. Many of these cases heal spontaneously without further surgery, but if acute symptoms do recur, drainage and curettement of the petrous pyramid usually relieve all symptoms, including the otorrhea.

Briefly we shall discuss three cases which illustrate the chronic phase of petrous suppuration.

REPORT OF CASES.

CASE 1.—A man, 42 years old, complained of intermittent earache in the right ear for about eight weeks, with only an occasional otorrhea. The x-ray examination showed average sized pneumatic mastoids of the adult type, made up mainly of small cells. The left appeared normal; the right was cloudy, with the cellular outlines ill-defined, although the trabeculæ could be faintly seen. The roentgenologist stated that from a radiographic standpoint there was no evidence of cellular necrosis, but it had the appearance of an acute mastoiditis. The petrosæ were pneumatic and clear.

A simple mastoidectomy was performed, and after three weeks of normal convalescence he complained of severe pain over the right eye and had a profuse discharge from the external auditory canal. The report of the x-ray examination at this time was as follows:

"The right mastoid shows the result of operative procedure with complete removal of cells in the mastoid portion of the temporal bone. The petrous area is opaque. As one compares the petrous pyramids, it is observed that there is a generalized diminution in aeration of those cells which lie at the base of the right pyramid. No cells are outlined toward the tip of the apex. The right pyramid shows moderate cloudiness, but there is no evidence of bone atrophy or decalcification such as is associated with suppurative petrositis. The appearance presented by the right pyramid is similar to that accompanying any inflammatory process of the mastoid."

At this time, four weeks after the primary operation, a secondary simple mastoidectomy was performed, with no relief of symptoms. Four days later the eye examination showed lateral rotation on the affected side slightly impaired.

One week after the secondary mastoidectomy an x-ray report read as follows: "In comparing these radiographs with those taken previously, it is clear that the appearance of the right petrous apex has been definitely changed. It appears hazy and has lost in part the definition of the border; the left apex remains clearly defined. It seems justifiable to diagnose a right petrositis at this time."

Three weeks after the secondary mastoidectomy all symptoms except the otorrhea had disappeared. Several months have now elapsed, and the last report

concerning the patient was that the ear was still discharging, but the discharge was decreasing in amount.

CASE 2.—A boy, 10 years old, had an acute earache, a headache, and a pain over the right eye, associated with a profuse aural discharge. These symptoms lasted for about ten days and then disappeared except for the otorrhea which had persisted for several months. Three days before consulting an otologist he developed an unbearable pain over the right eye, a severe headache, a temperature of 103, and double vision. A simple mastoidectomy was performed immediately; a complete external rectus paralysis and generalized meningitis followed, and the patient died. No x-rays were taken, and a petrous apex infection was not suspected by the surgeon in charge. This is obviously a neglected case and was one in which an apparently adequate petrosal egress became obstructed.

CASE 3.—A woman, aged 22, had an acute otitis media, followed four weeks later by a simple mastoidectomy, which did not heal. Following this, she had occasional first division pain, headaches, and a slight afternoon temperature. Six weeks later a secondary simple mastoidectomy was performed, without affecting either the pain or the otorrhea. Gradually, however, all symptoms disappeared except the otorrhea, and she appeared well. This persisted for several weeks, and one would assume the acute quiescent period to be over and a definitely adequate fistula to have been established. Certainly the picture would justify the clinical diagnosis of a chronic petrous suppuration.

This case came to my attention three or four months after the onset of the symptoms. She had severe first division pain and headache that could only be controlled by morphine, a septic temperature varying between 99.2 and 104, a definite external rectus paralysis, and a questionable Kernig sign. X-ray examination showed an incomplete exenteration of the mastoid cells and a petrous pyramid with cloudiness suggestive of complete destruction. Lipiodol injected into the pyramid passed through a perforation and lay in the base of the cranium. Drs. Kopetzky and Almour operated on this patient, and she made a complete recovery.

CONCLUSIONS.

1. A chronic petrous infection is clinically an extended quiescent stage of an acute petrositis.

2. Adequate drainage through a petrosal fistula explains the disappearance of the acute symptoms.

3. Many of these cases heal spontaneously.

4. A return of symptoms indicates obstruction in the fistulous tract which usually requires immediate operative interference.

5. X-ray studies are necessary in determining the anatomic structure of the petrosa, in estimating the amount and character of the destruction, and in ascertaining whether or not the bony confines of the petrosa are intact.

277 ALEXANDER STREET.

IV. THE FUNDAMENTALS OF THERAPY.

CXIII.

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My discussion of this subject has been simplified for me by the President's suggestion that four questions be considered.

1. "Are cells in the pyramidal portion of the temporal bone always infected in association with acute purulent otitis media or acute mastoiditis, and, if this is so, do not the great majority of all pyramid infections clear up after paracentesis or a simple mastoid operation?"

My answer to the first question is that neither the cells in the pyramidal portion nor the cells in the mastoid portion of the temporal bone are involved to considerable extent in every case of acute purulent otitis media. But in every case in which infection has become established in the mastoid cells it will be found to involve to a very considerable extent those cells of the pyramid that surround the semicircular canals, and those that lie between the posterior semicircular canals and the dome of the jugular bulb and those internal to the fallopian canal.

Resolution takes place in these pyramidal cells in the majority of cases, but in a number worth considering the infection in them will be the cause of delayed healing, secondary mastoid operation and at times serious complications, which might have been avoided by drainage of these cells at the first operation.

In a patient with a sclerotic mastoid process and a chronic purulent otitis media there is often little involvement of the mastoid but marked involvement of the cells at the base of the petrous pyramid and of the cells in the inner tympanic wall below the promontory and in front of it near the eustachian tube. Through these cells infection can travel into the petrous apex independently of infection in the mastoid cells. In radical mastoid surgery delayed healing and prolonged discharge, often ascribed to an open eustachian tube, may be due to infection in these cells. The fact that many simple mastoid wounds heal without the removal of the deeper cells does not justify

leaving these cells to jeopardize even a small percentage of cases when they can be safely removed.

2. "If a patient has an acute mastoid infection and a palsy of the sixth nerve on the same side, do these symptoms justify a radical operation at once, or is it advisable to widely open the drum and mastoid and await results?"

Mastoiditis associated with paralysis of the sixth nerve alone has not, in my experience, justified radical exenteration of the petrous apex; neither does acute otitis media associated with palsy of the sixth nerve always necessitate operation on the mastoid, as is illustrated by the following case. A girl of fourteen had an acute purulent otitis media of a few days' duration, during which paralysis of the sixth nerve developed on the same side. To all appearances, x-ray findings, etc., she had a mild middle ear infection for which myringotomy alone was indicated. No other operation was performed, and as the otorrhea ceased within a week and the palsy disappeared a few days later, the patient was discharged from the ward but was kept under observation for several months in the clinic. She was seen as late as a year afterwards and had had no recurrence of symptoms. In my experience sixth nerve palsy[1] has occurred so often in mastoiditis complicated by lateral sinus and jugular bulb infections without evident involvement of the petrous apex, that I am suspicious of jugular bulb involvement when sixth nerve palsy is observed. I was influenced by this sign in one case to investigate the sinus when other evidences of sinus involvement were not marked and was justified in doing so by finding a partial thrombosis of the jugular bulb. But when sixth nerve paralysis occurs after a simple mastoid operation in a patient with headache and but moderate fever it is sufficiently significant of deep cell involvement to warrant reoperation on the mastoid.

3. "If this more conservative treatment is followed, what symptoms do you watch for that would indicate the necessity for more adequate drainage of the pyramidal cells?"

Headache associated with delayed healing of the mastoid wound, or prolonged discharge from the middle ear with or without sixth nerve paralysis, calls for investigation of the mastoid with particular attention to those cells around and below the semicircular canals in the petrous pyramid. When these symptoms persist after a radical mastoid operation, the cells on the inner tympanic wall, below and in front of the promontory, should be examined, in addition to those near the semicircular canals.

4. "In other words, what are the points in the history, symptoms, the findings on x-ray and other methods of examination that indicate the *immediate radical operation* of Kopetzky, Eagleton and others? What proportion of patients will develop a serious or fatal complication as a result of being conservative, in the sense that a paracentesis and a simple mastoid operation is conservative treatment?"

Only in exceptional cases is the radical procedure of Eagleton,[2] Kopetzky[3] and Richards[4] indicated or intended, but the points that more often call for investigation of the cells referred to in the petrous are principally those mentioned above, namely, headache with prolonged discharge from the middle ear or the radical mastoid cavity, or headache with delayed healing of the mastoid wound. With these symptoms there may or may not be a sixth nerve paralysis, positive x-ray findings in the petrous pyramid, eyeground changes and various neuralgias. Persistent headache and discharge alone is sufficient to warrant reinvestigation of the wound and exploration of the deep cells. Experience has influenced me to be guided by the findings in each individual case and to follow gingerly and carefully the diseased structure deep into the petrous pyramid along the cells mentioned. In the cases that I have encountered, whatever infection there may have been deeper in the pyramid seems to have resolved after these cells have been removed, for the symptoms have subsided and the wounds have healed. One case that died of meningitis fifteen years ago after a simple and then a radical mastoid operation is perhaps an exception. My experience, I repeat, has been limited to the less severe types in which recovery occurred after exenteration of the cells around and below the labyrinth that may communicate with cells deeper in the apex. One other exception will be referred to later as a case of osteomyelitis of the base of the skull.

The kind of operation to be adopted depends on the severity of the symptoms, the type of case and the degree of involvement found in the mastoid and tympanum. If the symptoms persist following a radical mastoid operation on a patient with chronic purulent otitis media, but a normal labyrinth, and further operation is indicated, it seems safer to me to investigate the tympanic cells on the inner wall below and in front of the promontory with small curettes and a probe, as Bowers[5] did successfully in his case, with the hope of opening into a tract through which drainage may take place, rather than to use the burr, as Kopetzky[3] advises, notwithstanding his explicit measurements and directions. When the burr is used

along the route he describes in order to avoid the carotid and cochlea the danger of entering the dura where it dips into the hiatus fallopii seems to me to be a real one. When the labyrinth is dead and the severity of the symptoms warrants a more radical procedure, a modification of the operation described by Richards,[4] in 1927, in his report of eight cases would be my choice.

As to "What proportion of patients will develop a serious or fatal complication as a result of being conservative in the sense that a paracentesis and a simple mastoid is conservative treatment?"—this would depend entirely on the type and severity of the cases involved. The simple mastoid operation, as it was performed quite generally some years ago, would be of no more use in the presence of deeply involved cells than would a paracentesis, but a simple mastoid operation with exenteration of the deep cells around the labyrinth would probably in most cases avert the necessity of the more dangerous operation.

The radical removal of the apex may have to be undertaken when the *pathology indicates* it after the less dangerous exenteration of the cells approaching the apex has been performed without relief of symptoms or the discovery of a sinus tract leading to the deeper cells. The extensive operation, I think, applies only to cases with malignant involvement of the pyramid, and to the type with advanced necrosis and epidural abscess due to suppuration associated with dead labyrinths in cases of chronic suppuration, such as Richards[4] reported. An approach through the internal or intrapyramidal route seems preferable. I think the operation is rarely, if ever, indicated in cases with an active labyrinth. In acute cases with active labyrinths in which the deep cells have been removed without relief of symptoms, and in complicated cases with osteomyelitis and cortical perforation at the apex that Eagleton[2] reports, the approach may be better by the temporal route. But I cannot speak from experience with these cases.

Dwyer[6] also calls attention to the two different types of petrositis that occur with acute mastoid infection: Those that occur in pneumatic bone from infection in the middle ear and mastoid, and those that arise from infection in the nasopharynx, or from severe involvement of the sphenoid sinus are essentially osteomyelitis of the base of the skull. I have seen such a case at autopsy where the basilar process and the condyles of the occipital bone were softened by disease. Such cases are, as he says, usually "doomed from the start," and a very radical procedure may be justified. The more common

type of petrositis is that found in cases with pneumatic pyramids. This type will occur much less frequently when surgery for acute mastoiditis becomes generally more thorough. Interference with the lining membrane of the mastoid antrum and removal of the cells around the semicircular canals was discountenanced by some otologists not so long ago, and some of us remember how severely Blackwell was criticized before this Society for removing these cells and widening the aditus in his simple mastoid operation. Since then it has become more generally the practice to look for softened structure in this region and to follow it to its depth, which explains the fact that men whose technic is thorough in this respect tell me that they rarely see complications at the petrous apex.

127 EAST 62ND STREET.

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CXIV.

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CLEVELAND.

Neither pneumatization nor infection in the petrosal pyramid can in any way be associated with the great depression starting in 1929, and it is only fair to assume that either consciously or unconsciously we have been dealing with some form of infection in this region for about as long as we have been dealing with acute infections of the middle ear or mastoid, but as Eves has well said, "Previous to 1929, very few cases were diagnosed before autopsy."

All bone is capable of resisting a certain amount of infection but that containing red bone marrow, which consists of myeloid tissue and plays a rôle in hemopoiesis, is more resistant than that of the pneumatic type. Obviously it is important to understand the

*This paper was prepared by Dr. Mullin before his death, and was read by the Secretary of the Society.

character of the osseous structure or cellular development in the petrous pyramid if one wishes to thoroughly understand this infectious process. I have had the privilege of examining the splendid collection of temporal bones prepared by Dr. H. N. Glick of St. Louis. He demonstrated to me that in the majority of his specimens there exists a cellular communication between the middle ear, the mastoid, and that portion of the petrosal pyramid anterior to the cochlea. The whole group of cells may become infected during the same acute process and may all recover in exactly the same way.

As Carmack has said, in the presence of otitis media, there is every anatomic reason for an extension of the inflammatory process into the cells of the pyramid similar to that into the mastoid process. Therefore, I believe that petrositis is a descriptive term which is as applicable as is the term mastoiditis.

Dr. Glick's specimens also show that a well pneumatized mastoid is not always associated with a well pneumatized petrous pyramid. This fact is also corroborated by the late Dr. Ziegelman, who said, "The cellular character of the mastoid is not a true index to other portions of the temporal bone."

When dealing with a case of acute otitis media of a highly septic type, an x-ray study of the cellular development in the mastoid and petrous bone should be made as soon as possible. This is routine with us and is the so-called base plate referred to by Kopetzky, Coates and others. Orbital and retro-orbital pain coming on at any time during this acute stage should arouse suspicion of infection developing in the petrous cells. Continued pain, continued fever, continued aural discharge, or a reappearance of aural discharge after it has ceased, all indicate the necessity for further study. Roentgenograms of the petrous bone should be taken in different positions to show the pyramid at different angles; in this way we gain as much information as possible of the progress in cellular obliteration or bone changes. The combined x-ray findings must be correlated with the clinical course. That rare quality, surgical judgment, must determine the course to follow rather than dependence upon any dogmatic rules.

In defining treatment, myringotomy and complete simple mastoidectomy are conservative measures, while radical mastoidectomy and some surgical approach to the petrous apex are considered as radical measures.

Granting that an adequate myringotomy has been done without relief of symptoms, then a complete simple mastoidectomy should be

performed, referring repeatedly to the roentgenograms so that no remote cells are overlooked. Following this and barring immediate meningeal signs, time should be given to determine what results may follow the complete simple mastoid operation.

Should the symptoms progress rather than ameliorate, it is now time to do a radical mastoidectomy, making such an exposure that if a fistulous tract is present, it can be located and followed, or a suitable area prepared for surgical approach to the petrous tip.

Should there be merely discharge without pain, meningeal signs or perilabyrinthitis in an individual whose hearing was normal previous to the acute middle ear infection, then the important structures in the middle ear should not be sacrificed by radical operation without serious consideration of the above factors, and until a sufficient period of time has elapsed to measure the results of a less radical procedure.

Before the radical operation is performed it is necessary to differentiate between Gradenigo's syndrome and infection of the petrous apex. I heard Dr. Kopetzky discuss this subject lucidly and completely before the Pacific Coast Oto-Ophthalmological Society. He has given us some very definite statements, namely, "No diagnosis should rest solely on roentgen film findings." And again, "Every other lesion which could cause similar symptoms must be eliminated to establish the facts which make the diagnosis," and I would add to this latter statement that it applies very definitely to sixth nerve paralysis. And lastly, he said, "All cases of petrosal infection are not similar; hence, no one procedure is applicable to all."

I feel that he might well have confined his words of wisdom to the above statements, since it is not so easy to agree with that said in a later article in collaboration with Almour, namely, "With the arguments which are advanced to advocate limiting of surgery to simple mastoidectomy we have little sympathy. The place for so-called conservatism is not scientifically grounded."

Our series is limited to seven cases, six of our own and one seen in consultation. One case, that died of meningitis, I feel could have been saved if we had known as much about this condition then as we now do. The case seen in consultation I feel might have been saved from meningitis by an earlier complete simple mastoid operation. After a complete simple mastoidectomy in which all the cellular ramifications were followed, one patient had a temporary facial paralysis which later fully recovered. Her mastoid wound, however, discharged for forty-eight days. It eventually healed and has been

well for four years. Another case, a growing boy with a large cellular mastoid and acute mastoiditis, has no symptoms of petrous infection other than positive x-ray findings and slight discharge from the mastoid wound following operation, and is still under observation. His mastoid has been thoroughly cleaned out and we feel very definitely that his wound will heal. The point I make is that three cases have made complete recovery following a complete simple mastoid operation during the acute stage. They required patience and long after-treatment. Two other patients required radical operations and two died.

CXV.

ISIDORE FRIESNER, M. D., AND

J. G. DRUSS, M. D.,

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The early literature, our own clinical experience and the more recently reported experiences of Voss, Baldenwick, Ruttin, Sears, Perkins, Neumann, Coates, Ersner and Myers, and many others lend support to our belief that suppuration confined within the petrous pyramid has a marked tendency to heal spontaneously. This fact is based, obviously, upon such widespread clinical experience that it is patent that the presence of suppuration in the petrous pyramid is not, *per se*, an indication for immediate operation upon that structure. This phase of the subject, viz., spontaneous healing, has been so thoroughly presented that we prefer to spend the time allotted to us in a discussion, chiefly, of the fundamentals of surgical therapy.

In this, as in other diseases, anatomy and pathology constitute the very foundation of all surgical procedures, and therefore, we deem it timely, at this juncture, to present the results of our studies along these lines. Our investigations have been carried on for the past seven or eight years and consist, in part, of the histologic examination of twenty-four temporal bones of patients who died of intracranial complications of otitic disease. Of the twenty-four cases, thirteen had intracranial lesions when admitted to the hospital, nine of the latter had meningitis and four had brain abscess. The material is almost exclusively from cases with acute symptoms.

Before presenting our histopathologic findings in cases of suppuration of the petrous pyramid, it is of considerable importance to emphasize what we consider to be certain basic facts. Petrous pyra-

mid suppurations are frequently found histologically in patients who died of otitic intracranial complications. In some of these patients there were no clinical evidences of petrositis at any time, nor any basis for suspecting that such a condition existed. The pathologic findings were discovered quite by accident in the routine examination of our material. Even in retrospect, it is at times difficult or impossible to correlate the clinical with the histologic findings. Nevertheless, the fact remains that there were cases of petrositis (histologically) which insofar as we were able to ascertain, gave neither typical clinical signs nor symptoms. In the early stages of acute suppuration of the middle ear, it is well known that there may be associated involvement not only of the pneumatic cells in the mastoid, but also of the cells in the petrous pyramid. This inflammatory involvement may take place simultaneously with the onset of the otitis media or may follow shortly thereafter. These changes in the pneumatic spaces may at this stage vary in degree from thickening and infiltration of the mucosa to an extravasation of purulent exudate, and may be present without characteristic signs or symptoms.

In the consideration of petrositis it is important to include pathologic changes which occur in the perilabyrinthine structures as well as those that occur in the apex. Cases of perilabyrinthitis and deep extradural abscess, which undoubtedly should be considered as petrositis, have been reported in the literature. It must always be kept in mind that the lesion in the petrous pyramid does not extend to the apex in all cases. Nor are the symptoms dependent upon an extension of the lesion to the apex. We have repeatedly called attention to these facts, and we consider them of fundamental importance as related to the therapy of petrositis.

The histologic diagnosis of petrositis was made from the study of the serial sections of twenty-four temporal bones; for twenty of these, the sections include the extreme tip of the apex. With regard to anatomic structure: the mastoid process was pneumatic in twelve cases (50 per cent), mixed in nine (37½ per cent) and diploetic in three (12½ per cent). The petrous pyramid was pneumatic in four cases (16⅔ per cent), mixed in eighteen cases (75 per cent), and diploetic in two cases (8⅓ per cent). Of the twenty cases in which the apex was sectioned, it was pneumatic in one case (5 per cent), mixed in six cases (30 per cent) and diploetic in thirteen cases (65 per cent).

It must be borne in mind that in classifying temporal bones as to type of anatomic structure, no definite criteria have been established, and that the personal interpretation of the observer, therefore,

is an important factor. The age of the individual must also be taken into account in considering the extent of pneumatization of the temporal bone, insofar as the completion of pneumatization of the petrous apex takes place later than it does in the mastoid. Of the twenty cases in which the apex was sectioned, ten (50 per cent) of the patients were under ten years of age. This perhaps may account for our large percentage (i. e., thirteen out of twenty) diploetic apices.

While we are aware that no definite conclusions can be drawn from so small a series, the findings, nevertheless, are extremely suggestive, inasmuch as the above data were obtained from the study of histologic sections. The chances of error are thus diminished. The figures obtained from the literature up to the present time have been based mainly upon gross observations made on unsectioned temporal bones.

According to the above figures, extensive pneumatization in the mastoid is present much more frequently than in the petrous pyramid and the apex. However, in isolated instances the pneumatization in the petrous pyramid may be more advanced than in the mastoid process. Partial pneumatization (mixed type) is more frequently found in the petrous pyramid than in the mastoid bone. This may be accounted for by the fact that the process of pneumatization extends from the tympanic cavity and eustachian tube into the petrous pyramid and apex and may continue after pneumatization in the mastoid has terminated.

As stated previously, the greater the degree of pneumatization in the temporal bone, the greater is the facility with which infection spreads through it. In the majority of cases, infection spreads into the petrous pyramid from the tympanum and antrum along the perilabyrinthine cells. None but an arbitrary division between these various perilabyrinthine cells can be made. The infectious process as it advances frequently involves adjacent marrow spaces and the more dense bone. In the strict sense of the term, an osteitis alone is rarely present. Instead, the picture is often that of a combination of osteitis and osteomyelitis.

Since there are no definite lines of demarcation between the various groups of perilabyrinthine cells, and since extension may take place along more than one route, it is at times difficult, even by careful histologic study, to determine the exact pathway of extension. In the instances in which infection involved the entire perilabyrinthine group of cells or a considerable portion of them, we

have identified the pathway through which the infection extended, by locating the area in which the maximum inflammatory change and destruction occurred.

In nineteen of the twenty-four cases the infection spread from the tympanum and antrum along the superior and posterior perilabyrinthine structures. In some of these cases, however, the infralabyrinthine cells were also involved. The supralabyrinthine route of extension was the predominant one in fourteen of the cases. The infralabyrinthine route was the predominant one in two cases. The infectious process in part extended through the fossa subarcuata in two cases and in one of these the latter was the predominant pathway. In three cases the extension took place mainly along the peritubal cells. In two cases the infection extended along the carotid canal. In two cases, the route of extension could not be determined with any degree of accuracy.

The postero-superior route, we found, was by far the most frequent. It cannot be stated too emphatically that lesions in the petrous pyramid do not always extend to the apex. In the majority of our cases the greatest expression of the disease process was noted in the petrous pyramid between the superior semicircular canal and the internal auditory meatus. Furthermore, in fourteen of the twenty-four cases, the lesion was most manifest at the superior posterior margin of the petrosa. While it is true that the lesion in the petrosa may extend into the apex, the pathologic changes at that site are usually not so marked as in the lateral portion of the pyramid. In eleven of the twenty apices sectioned we found moderately severe inflammatory changes. In four there were slight changes and the findings were relatively normal in five. However, in only three of the cases were the inflammatory changes in the apex more marked than in the remainder of the pyramid. The duration of the infectious process may be a factor in the extent of the involvement of the petrosa and apex. As is well known, the suppurative process frequently erodes the bony cortex of the petrosa, along the middle or posterior fossa at an appreciable distance from the apex, and produces an extradural space. These changes may take place before any considerable degree of involvement of the apex has occurred.

In eleven of the twenty-four cases there was either a frank extradural abscess or a communication between the suppurative process in the bone and the adjacent overlying dura. The most significant symptom of these epidural abscesses was pain; and in ten of the eleven cases of epidural abscess the patients complained of severe headache. The inflammatory process involved chiefly the dura

of the middle fossa in seven cases, and that of the posterior fossa in four cases. In a few of these, there were multiple perforations of the bony cortex of the pyramid in the middle or posterior fossa, or both. The inflammatory changes in the dura varied in extent, remaining localized in some instances, and in others extending to the apex.

It is obvious from our material that there are no symptoms which are invariably associated with disease of the petrous pyramid. The most significant symptom is pain. In our clinical cases the subsidence of pain marked the beginning of recovery. On the other hand, the pathologic material demonstrated, in all but one instance, that pain persisted when the suppurative process in the petrous pyramid had perforated the cortex and involved the dura.

The generally accepted belief that serious disease in the petrous pyramid is always associated with persistent or recurring discharge from the tympanum is also erroneous. A scant discharge when associated with pain, is of much graver import than a profuse discharge. Heretofore, insufficient stress has been laid on the type of case in which pain develops following an acute otitis and an operated mastoiditis, the middle ear and mastoid resolving and healing. In our experience this condition is always extremely serious. In an earlier communication we demonstrated histologically that in cases of osteitis of the pyramid, inflammation of the superficial structures (i. e., middle ear, antrum, etc.) may resolve and heal, yet virulent infection with progressive destruction may continue in the depths of the petrosa. We have encountered instances also in which, following an acute suppurative otitis and symptoms of mastoiditis, operation disclosed little or no gross disease in the mastoid. In these cases not infrequently the middle ear undergoes rapid resolution and the mastoid wound heals. The hearing may be excellent. The persistence of severe pain, with or without fever, demands immediate surgical intervention. In this type of case we have seen, both at operation and histologically, the most extensive destruction in the pyramid. Occasionally, too, an external rectus palsy appears and must be considered a focal sign.

Osteitis of the pyramid may occur as a complication of an acute suppurative otitis media and an acute suppurative mastoiditis. Under such circumstances, shortly after the mastoid operation, there occurs a profuse discharge from either the middle ear, the mastoid wound or both. We have been conservative in this type of case because the bone focus obviously was draining. We have demonstrated that the discharge contains the products of bone disintegration; at times as much as 37 mgms. of calcium per 100 cc. is present (Friesner and

Rosen). In some instances profuse discharge persisted for four or even eight weeks. We have seen roentgenographic evidence of changes in the petrous pyramid in the presence of a completely exenterated mastoid, yet the absence of high fever, the gradual diminution of pain and the lack of evidence that an intracranial extension was imminent led us to persist in our conservative course. Most of the cases in this group recovered without further surgical measures. Furthermore, it is most important to add that the end result in these cases, even after eight weeks of profuse discharge, was a normal middle ear, normal hearing and a healed mastoid wound.

We have learned from our pathologic material that in the vast majority of cases suppuration in the pyramid extended from the middle ear and mastoid antrum through the superior and posterior group of perilabyrinthine cells. Secondly, we found the greatest expression of disease at the posterosuperior margin of the petrous pyramid between the superior semicircular canal and internal auditory meatus. In only a few cases was the major expression of the disease at the apex of the pyramid. As has been stated before, all the clinically observed cases which recovered have healed with normal ears and normal hearing.

We have based our concept of surgical therapy upon the aforementioned facts, choosing a type of operation which we find gives adequate exposure of the region in which disease is most apt to be found. Secondly, the necessary surgical measures can be performed with good illumination, under direct vision, thus enabling one to conserve the important structures in the petrous pyramid.

In operations on the pyramid we have attempted to base our indications upon our pathologic, clinical and operative experiences. The indications in some instances are, according to our present views, clear cut. In others, each case must be judged individually, using all possible criteria, including at times a study of the cytology of the cerebrospinal fluid. Here, too, due consideration must be given to the time relationship between the onset of the otitis and the development of symptoms indicative of petrosal disease. As a general rule, the later the onset of symptoms indicative of disease in the pyramid the graver their import.

The cases presenting indications for exploration of the pyramid may be divided into two groups:

1. *Cases in which no mastoidectomy has been performed.* (A) In cases in which the ear has been discharging for several weeks; when pain has persisted or become increasingly severe, particularly

when to the original pain in the eye, the teeth, or the ear, there is added severe frontal headache, exploration of the pyramid should immediately follow mastoidectomy, if the findings in the mastoid are considered inadequate to explain the pain. (B) In cases in which the ear has been discharging for several weeks, with no relief of pain, if meningeal signs (meningitis sympathica) develop, and the operative findings in the mastoid are inadequate to explain the intracranial extension, exploration of the pyramid should be carried out immediately following the mastoidectomy. We realize, of course, that it is extremely difficult to accurately paraphrase the term "adequate findings to explain pain." In the final analysis, the personal interpretation of the surgeon must be paramount. It might be timely, however, to reiterate that we have seen headache, pain in eye, fever and persistent otorrhea from an undrained zygomatic abscess; fifth and sixth nerve involvement from an epidural abscess over the mastoid tegmen; meningeal symptoms from a perisinus and epidural abscess or sinus thrombosis. These are some of the findings in the mastoid that we would consider adequate to explain pain. Of course a badly and extensively diseased mastoid is another. Shambaugh reported temporal headache and external rectus paralysis as the result of a small epidural abscess in the root of the zygoma, in the presence of a resolved ear and healed mastoid wound.

2. *Cases in which mastoidectomy has been performed.* (A) In some of the cases of acute otitis with bone involvement, the major expression of the inflammation is in the pyramid rather than in the mastoid. Not infrequently in such a patient, because of continued pain, profuse otorrhea, fever and tenderness, the mastoid is operated upon and discloses little or no pathologic change. The middle ear may resolve rapidly and the mastoid wound heal promptly. As we have already stated, the most virulent and most serious cases of osteitis of the petrosa are those in which the middle ear is dry and the mastoid wound almost or entirely healed. In these, if severe pain persists or begins late, and particularly if focal signs (sixth, seventh, or eighth nerve involvement) develop, operation is imperative. (B) In our clinical experience, by far the largest group of cases are those in which, following mastoidectomy, disease in the pyramid manifests itself by pain and the development of increasing discharge from the middle ear, the mastoid wound or both. As regards indications for operation, this group offers the most difficult problem. All diagnostic criteria should be used in this type of case. There can be no question that many such cases recover spontaneously. Yet if at any time, in the judgment of the surgeon, such a patient is not doing

well and an intracranial extension seems imminent, the pyramid should be explored.

CONCLUSION.

Correlation of our anatomic and histopathologic studies with our clinical experience, makes evident certain facts which are of fundamental importance in the therapy of petrous pyramid suppuration.

1. That suppurations in the petrous pyramid tend to drain and heal spontaneously.

2. That by no means does the lesion in the petrous pyramid in all cases extend to the apex. Nor are the symptoms dependent upon extension of the lesions to the apex.

3. That the posterosuperior route is the frequent pathway of extension and that the greatest expression of the disease process is noted in the area between the superior semicircular canal and the internal auditory meatus, at the posterior superior margin of the petrosa.

4. That in our clinical experience the cessation of pain, in all but one instance, marked beginning recovery.

5. With the above facts in mind, we have established certain indications.

6. It is obvious that no one procedure is adequate to drain all foci in the pyramid.

CXVI.

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The apparent differences of opinion between the various writers on the subject of petrositis have, in the main, been confined to the therapy indicated for the cure of the lesion. The diagnostic picture has been almost universally accepted. Yet, if one critically studies the several views dealing with the therapy of this lesion, one is impressed with the fact that each is true to a certain extent. Wherein, then, do the discrepancies lie? The author is certain, after a thorough perusal of the literature, that the opinions of the various workers

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are separated only by the fact that most of them have seen and treated only one, or at the most, a few types of petrositis. In a paper which is now in publication the author has advanced the suggestion that in the lack of a proper nomenclature lies the difficulty in the way to a harmonious agreement of all investigators.

The nomenclature proposed is based upon anatomic and clinical facts which are evident from the study of the cases reported.

Since the symptomatology has already been discussed and the finer points in the differentiation of these several varieties of petrositis have been presented, it remains only to outline the nomenclature which, the author feels, is the key to the fundamentals of therapy in suppurations of the petrous perilabyrinth.

NOMENCLATURE.

1. Posterosuperior petrositis.
2. Postero-inferior petrositis.
3. Postero-internal petrositis.
4. Anterior supracochlear petrositis.
5. Anterior subcochlear petrositis.
6. Anterior precochlear (pericarotid) petrositis.
7. Apicitis (empyema of petrous apex).

The determination of whether the lesion is acute, subacute or chronic depends upon the clinical findings, and any of the above anatomic designations may be prefaced by the clinical term of acute, subacute or chronic.

Utilizing this terminology, it at once becomes evident that all lesions of the perilabyrinth are not amenable to any one type of therapy. What suffices for a cure of an acute posterosuperior petrositis will not be effective in the management of an acute anterior subcochlear lesion. It also becomes evident that the attempt to classify the therapeutics of petrositis into conservative and radical is an error which must not be continued. The surgical approach to a postero-internal petrositis is necessarily different from that for an apicitis, and each is conservative for the respective lesion but radical for the other.

The determination of the type of petrositis and its location in the perilabyrinth is possible in the greatest number of cases by placing proper emphasis upon the minutiæ in the clinical picture. All varieties of petrositis present the characteristic retro- or peri-orbital pain and tympanomastoid suppuration; the acute and subacute lesions present,

in addition, a low grade sepsis. It is the other details in the clinical picture which tell the otologist the exact location of the lesion. Once the location is noted the therapeutic indication becomes self-evident.

LESIONS CONFINED TO THE POSTERIOR PERILABYRINTH.

These are the easiest to manage. The milder cases respond to the drainage afforded this area by a thorough, simple mastoidectomy. The posterosuperior petrositis will present a fistula in the area located above the horizontal semicircular canal and behind the superior semicircular canal, which at times may lead directly into the petrous apex. The management of this type consists in an enlargement of the fistula and a thorough probing and curettage of the entire tract, followed by saline irrigation and the injection of a contrast substance, preferably lipiodol. This procedure effects a cure in most cases. When postoperative symptoms continue beyond a reasonable length of time, however, one then reasons that the disease has spread from its original site in the posterosuperior perilabyrinth into the apex and that further surgery is necessary.

The postero-inferior lesions show a fistulous tract located in the area below the posterior semicircular canal which should also be handled in the manner just described. Continuation of symptoms calls for further surgery.

The postero-internal lesions are the severest of this group, in that they extend toward the internal auditory meatus and threaten an early meningitis by rupture at this point. They are best managed by a complete removal of the inner table of the mastoid process over the sinus and anterior cerebellar surface, a careful separation of the dura inward toward the internal meatus, and a thorough curettage and removal of all diseased structures in the posterior perilabyrinth, skeletonizing, if necessary, the semicircular canals.

LESIONS CONFINED TO THE ANTERIOR PERILABYRINTH.

These constitute a greater threat to life than do the posterior petrosites because, firstly, they are not demonstrable through the exposure afforded by the simple mastoidectomy and, secondly, because of this, the search for the focus is often delayed beyond the safety point, due to the unwillingness of the surgeon to perform, or the patient to submit to, the exposure of the tympanic cavity which is essential for the discovery of the lesion.

Anterior supracochlear petrositis can often be adequately managed by the exposure afforded by a simple mastoid operation extended to expose the area in front of the superior semicircular canal. The

demonstrable fistula should either be managed as before mentioned or, if extension has occurred, the conversion of the tract into a trough extending to the apex, as recently described by Myerson, will very often suffice. When these procedures fail to result in the subsidence of the symptoms, a thorough exposure of the middle ear is called for, and an intrapetrosal drainage of the apex is imperative in order to afford drainage at the most advantageous point.

Anterior subcochlear and precochlear petrositis require the opening of the tympanic cavity in order to bring the lesion to view. The management of the fistulæ found is no different than that already described.

We now come to the variety of petrositis which we prefer to term apicitis or empyema of the petrous apex. The Germans term it Tiefgelegener Abscess im Felsenbeinspitze. The French term it Osteite profunde du pointe du rocher. It is for this area of the perilabyrinth that special technics have been devised and wherein a special technic is needed.

The petrous apex can be likened to Rome, since all roads in the perilabyrinth lead to it. Most of the cases of apicitis fall into the subacute and chronic groups, all of which present a fistulous tract leading from some portion of the perilabyrinth directly into the apex. These have already been considered, and the cure of these lesions is brought about by the proper management of the fistulous tract. In the acute apicitis, however, no macroscopic opening into the apex is anywhere demonstrable and it is therefore expedient to attack the apex directly if the patient's life is to be spared.

The methods proposed for the direct attack of the apex fall into two groups, the extrapetrosal and the intrapetrosal. The former comprise the various subdural approaches to the apex, while the latter confine themselves to the drainage of the apex through the temporal bone. Each has its indication and each possesses advantages which the other has not. The determination of when to employ the extrapetrosal approach and when the intrapetrosal is a matter for careful consideration. It is hoped that the following analysis will serve to clarify the question.

THE EXTRAPETROSAL (SUBDURAL) APPROACHES.

All the procedures in this group are undertaken by separating the dura of the middle cranial fossa from the superior surface of the petrous bone. They are represented by (1) Separating the dura through the simple mastoidectomy exposure; (2) Eagleton's procedure, and (3) the Gasserian ganglion approach.

Collectively, these procedures offer:

1. Adequate drainage of an extradural abscess which has resulted from the rupture of an apicitis through the superior surface of the petrous bone.

2. The surgical exposure of the superior petrosal surface, with the surgical attack of the apex at its uppermost point.

Collectively, these procedures present the following limitations:

1. They are inadequate in draining posterior perilabyrinth lesions because they do not reach the lesion.

2. They will not reach an extradural abscess which has ruptured from the apex into the posterior fossa.

Individually, the subdural approaches present the following advantages and disadvantages:

1. The subdural approaches which deliberately avoid the performance of a radical mastoidectomy and attempt the elevation of the dura through the exposure afforded by a simple mastoidectomy are, except where an extradural abscess is found in the middle cranial fossa, inadequate and harmful procedures; because

(a) They do not permit the inspection of the inner tympanic wall for the presence of fistulæ into the anterior perilabyrinthine spaces.

(b) The elevation of the dura must, of necessity, be started behind the superior semicircular canal, and in this way the dural insertions into the subarcuate fossa and into the canals for the petrosal nerves are torn. In addition, the numerous vascular connections between the dura, the petrous bone and the tegmen tympani, as Batson has recently demonstrated, are severed, thus increasing the danger of an ensuing purulent meningitis.

(c) They are technically the most unsatisfactory of all approaches to the apex because the point of attack is further away from the objective than that of the other subdural operations.

2. The subdural approach through the extended radical mastoidectomy, as described by Eagleton, is a rational one in that it affords, before anything else is attempted, an inspection of the entire inner tympanic wall for the presence of a fistula.

(a) Since the dural elevation in this procedure takes place in front of the subarcuate fossa and the emergence of the petrosal nerves

the injury to the dura is minimal, especially since a large portion of the squama is removed to allow for cerebral displacement.

(b) Its only disadvantage is that it does not afford drainage for the pus in the apex at the most dependent point.

3. The Gasserian ganglion approach does not meet the indication for surgery to the petrous apex because it does not allow of an inspection of the inner tympanic wall for fistulæ. In addition, it presupposes that *all* cases of petrositis present a lesion located in the apex, which we know is not the case.

THE INTRAPETROSAL APPROACHES.

These are represented by (1) Ramadier's procedure and (2) the author's.

These are the operations of choice in acute apicitis because:

1. They afford a thorough exposure of all possible avenues through which an infection can gain entrance to the various regions of the perilabyrinth.

2. Except where a rupture subdurally has occurred, they adequately meet every indication for the eradication of an apicitis.

3. They keep the infection and its point of drainage within the temporal bone and away from the endocranium, thus reducing to a minimum the possibility of a subsequent meningitis.

4. They afford drainage at the most dependent portion of the apex.

From the above outline it is at once evident that no one operative procedure suffices for every case of petrositis. For the majority of cases of petrosal suppuration there is no need for the mastery of a special technic; the majority of the cases show either a fistula into one of the subdivisions of the perilabyrinth, which in a great many instances leads directly to the tip, or show a necrosis leading toward the internal auditory meatus located just anterior to Trautmann's triangle. The intelligent and deft use of a curette and the widening of the fistula by means of a burr, the evacuation of the pus by washing with saline, the curettage of the granulations within the perilabyrinthine cavity will usually effect a cure. In any event, the failure to locate a point of disease in the posterior perilabyrinth, the only part exposed by a simple mastoidectomy, calls for a radical mastoidectomy to enable the operator to inspect the anterior perilabyrinth.

It is only in cases of encapsulated empyemas of that portion of the perilabyrinth which embraces the apex of the petrous bone that a specialized technic is indicated. Whether the subdural approach is used to reach the apex or one of the intrapetrosal methods is immaterial, provided one is thoroughly familiar with the anatomy embraced by the operation, aware of the advantages and disadvantages of the various technics, and is thoroughly convinced before operating that the patient has an encapsulated empyema of the apex.

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CXVII.

WELLS P. EAGLETON, M. D.,

NEWARK.

HISTORICAL BACKGROUND OF SURGICAL TREATMENT OF PETROUS APEX INFECTIONS.

On December 8, 1926, there entered the Newark Eye and Ear Infirmary a young girl (A. R.),[1] with a running ear that apparently required a simple mastoid operation. Following the operation she had pain (a) in her head (b) behind her eye,[2,3,4,5] which subsided. Later she returned with slight fever; and as the ear was still discharging and she had pain in her head the mastoid was curetted, during which the surgeon noticed tissue filling the anterior part of the middle ear cavity that "looked like cheese."

Over a period of forty-three days, subsequent to the first operation, she had recurrent attacks of pain in the head and the eye. The mastoid was recuretted twice. She then developed meningitis. At the autopsy the bone of the petrous apex was found eroded and contained a cavity filled with granulations and caseous material without pus. The apical suppuration had ultimately given rise to a terminal hemorrhagic meningo-encephalitis. I concluded that the granulation mass must be tuberculous.

I reported the case and exhibited the pathologic specimen at the spring meeting of the New York Otological Society, in 1927, as "tuberculosis of the petrous pyramid."

Two months later, on February 18, 1927, another patient (J. McE.),[6] was admitted who had a similar history of pain behind the eye, a running ear and, in addition, a facial paralysis. After thirteen days' illness he also developed meningitis and died. When at the

autopsy there was found a similar condition of granulomatous caries —without pus—with perforation of both the superior and posterior surfaces of the apex, the compact bone of the superior angle of the apex remaining intact, I realized that both patients had suffered from suppurative disease of the petrous apex.

Both of these cases,[7] with original drawings of the pathologic specimens,[8] were reported at a joint meeting of the Otological Sections of the New York Academy of Medicine and the College of Physicians of Philadelphia, in New York, on April 27, 1927, with the statement that as these "cases had run protracted courses of thirteen and forty-two days, respectively, during which time there were signs that the bone lesion and the meningitis were localized, properly directed operative procedure upon the bone prior to the advent of the terminal meningitis should have resulted in the cure of the patient."

This paper was abstracted and read at Copenhagen on August 2, 1928.[9] The full paper with four personal cases and anatomic studies on the fifth and sixth nerves was submitted for publication on September 3, 1929.[10]

Anatomic Researches.—The summer of 1927 was devoted to anatomic dissections at the École de Médicine, in an effort to establish an anatomic route into the petrous apex which would allow adequate inspection of the diseased area. The method selected I termed "unlocking the petrous apex." It is a modification of the Krause-Harley-Cushing approach to the Gasserian ganglion enlarged posteriorly. It was described in a paper read before the Otological Section of the British Medical Society at Manchester in July, 1929.[11,12]

First Exploration for a Diagnosed Apical Suppuration.—The first operation in the history of medicine of deliberately entering the petrous apex upon a patient whose condition had been previously diagnosed as "suppuration within the petrous apex," after a preliminary ligation of the common carotid, was performed on June 30, 1929. The patient (B. L.)[13] recovered promptly, although the operation so aggravated the localized meningitis that micro-organisms were temporarily free in the cerebrospinal fluid.[14,15]

Previous Cases of Drainage of the Apex Following the Accidental Finding of a Fistula Leading Into the Apex.—Bircher,[16] in 1893, drained an abscess in the petrous apex by following a fistulous tract from the middle ear; the patient recovered. And Brockaert,[17] in 1914, successfully opened Meckel's cave for a localized meningitis

of otitis origin, associated with neuralgia of the face and paralysis of the abductor muscle. However, neither operator opened the apex.

Richards,[18] in 1927, described "the petrous pyramid, its surgical anatomy and the technic of the operation for its removal," following an exenteration of the labyrinth.

Several others had drained the apex by accidental finding of a fistulous tract opening into the middle ear or the mastoid.[19]

In December, 1930, Kopetzky and Almour[20] devised an operation for entering the apex from the tympanic cavity by a small opening with a burr. Since that time Ramadier[21] (1931) has suggested temporary ligation of the internal carotid after its evulsion from its canal in the pyramid; and Frenckner[22] (1932) and Ruttin[23] (1933) have described various operations in which they followed an existent pneumatic tract.

The present report should be regarded as a supplement to my communications on "Meningitis from the Sphenoid," and my discussion of Vail's paper,[24] published in 1932.[25,26]

PURPOSE OF PRESENT STUDY.

Inadequacy of All Nomenclature of Infective Osteomyelitis Which Attacks the General Skeletal Bones; and Lack of Differential Classification Descriptive of the Various Pyogenic Lesions of a Petrous Apex or the Sphenoidal Basis.—A search through the literature fails to reveal any classification which adequately differentiates between the various forms of osteomyelitic processes that attack marrow-containing bones of the skeletal frame, or the distinctive forms of infective osteomyelitic pyogenic lesions of the cranium.

And no nomenclature whatsoever had been published which even attempted to differentiate between the various infective processes that have their seat in the petrous apex, prior to the outline classification that I presented to the American College of Surgeons, at Boston, on October 19, 1934.[27,28]

Nomenclature Based on Pathology as Influenced by the Embryologic Origin of Bone, Which Dictates Not Only the Specific Morphological Structure of the Bone but Also Its Specific Reaction to Infection.—An adequate nomenclature of pyogenic diseases of the skull must be based, not only upon recognized lesions of bone pathology, as presented by the inflammatory, metabolic and neoplastic processes, but also as influenced by the peculiar embryogenesis of the particular bone attacked. For the mammalian cranium is composed of three

genetically different and specific types of bone which determine the particular pathologic form that the lesion will assume and the course that the disease will take.

Thus, (a) the embryologic origin of the bone, and (b) the stage of its growth at the time of its invasion by bacteria, are factors in osseous pyogenic diseases, the importance of which has not been appreciated, although they are now recognized as of commanding influence in the growth of bony neoplasms.

SURGICAL LESIONS OF THE APEX.

1. *Histopathology of Abscess of the Petrous Apex.*—In recent years the morphology of the apex of the petrous pyramid (Baldenweck,[29] Pietrantoni,[30] and Eagleton[15]) and the surgical anatomico-pathology of abscess within it, have been fully examined. (Eagleton,[10 15] Friesner and Druss.[31]).

(a) Invasive and (b) Quiescent Infective Osteomyelitis of the Petrous Apex Without Abscess Formation.—However, the literature contains but scant reference to the blood-borne infections which may attack the marrow within the bone of the petrous apex. These infections, during their most active stage, are without massive abscess formation, although they may have small foci of suppuration within them. This group of osteomyelitics of the sinusoidal spaces is of great surgical importance, since it is in this type of pyogenic inflammations that an operation upon the apex at a time when the patient was not profoundly sick, because the infection was semi-quiescent, precipitated (a) an invasive, (b) septic or (c) phlegmonous osteomyelitis accompanied by a rapidly fatal (d) meningo-encephalitis.

(2) Destructive and (3) Nondestructive Sinusoidal Osteomyelitic Phlegmons of the Sphenoidal Basis and of a Petrous Apex.—No attention has been directed to the unique histologic structure or the distinctive inflammatory patterns of the diffuse lesions that involve the spheno-occipital basis, with which the petrous apices are in close relationship. For these destructive but nonsuppurative infections of the basicranium I propose the generic designation "phlebitic sinusoidal osteomyelitic phlegmons."

CLASSIFICATION OF CLINICAL TYPES OF APICAL SUPPURATION.

I now offer an amplification of my previous clinical classification of apical suppurative diseases.[27] It is an attempt to differentiate the various lesions in patients under my care, correlated with the radiographic, operative, postmortem and microscopic findings.

For in infections of the passively protective, pneumatic, and the actively protective marrow-filled bones of the neurocranium a suitable nomenclature is necessary; and it is hoped that the following will be of assistance in the diagnosis of infective lesions of the petrous apex and of the sphenoid basis, as well as of osteogenetic affections of the skull in general.

CLINICAL TYPES OF APICAL SUPPURATION AND ASSOCIATED OSTEOMYELITIC LESIONS OF THE SKULL

(Based on 42 cases of apical suppuration and 24 cases of sphenoidal bone disease associated with meningitis.)

I. *Reactive, Reparative and Protective Types of Osteomyelitis, When Marrow Is Present, or Osteitis in Pneumatized Bone.*

1. Sclerosing osteitis (analogous to Garre nonsuppurative osteomyelitis)—largely a reparative process.

2. Reactive osteomyelitis (analogous to Ollier's albuminous noninfective periosteitis of the long bones).

3. Granulomatous plasma-cell osteomyelitic invasion of the mastoid from an infected petrous apex (analogous to Pott's "puffy swelling.")[32]

4. "Sclerosing osteitis" of the apex adjacent to the labyrinth, accompanying a *sequestration of the labyrinth*—"exfoliative sequestration of the labyrinth." This type is usually (a) tubercular, but it may be (b) purulent, especially in scarlet fever, or from (c) operative trauma.[33]

II. *Nonsuppurative Congestive Cases—The Apical Symptoms Being Due to Venous Stasis.*

Subacute congestive osteomyelitis (without suppuration) associated with venous stasis.[27]

III. *Chronic Bone Septic Cases (Without Abscess Formation).*

Some of the cases of the chronic bone sepsis that are without pus originate in (a) osteothrombophlebitis of an adjacent area, such as the mastoid or the lateral sinus; others are the result of (b) direct tissue suppuration within a tract of pneumatic cells that passes over or behind the labyrinth; while a few are (c) embolic in origin, the infecting nidus having been brought by the circulation from a distant focus.

1. (a) Subacute, (b) slow (lenta), or (c) latent "*focal cariogranulomatous osteomyelitis*," confined to the apex (without macroscopic pus.[13]

2. (a) Subacute, (b) slow (lenta), or (c) latent "*diffuse embolic osteomyelitis*," involving large areas of the temporal and adjacent bones.

IV. *Abscess of the Apex.*

A. *Cariogranulomatous Abscess of the Apex Without Evidence of a Tract Connecting It With the Tympanomastoid Area:*

1. "Extradural abscess in the middle fossa." A localized granulomatous abscess with a perforation of the cortex below the Gasserian ganglion.[34]

2. "Subdural space abscess of the posterior fossa." A localized abscess in the posterosuperior quadrant of the apex, which by perforating the cortex causes a subdural space abscess of the posterior fossa situated mesial to the internal auditory meatus.

3. (a) "Cariogranulomatous osteomyelitic mass filling the entire apex," with perforations into both middle and posterior fossæ with pus; (b) without free pus.[35]

4. "Encapsulated abscess in the apex," the result of (a) direct extension of infection through pneumatized cells, or originating in (b) sinusoidal space osteomyelitis.

B. *Petrous Apex Abscess and Perilabyrinthine Suppuration With a Tract:*

5. "Peritubal tract" in the region of the eustachian tube cells, leading into an apical abscess. It may be associated with (a) "localized perilabyrinthine granulomatous caries," or (b) "perilabyrinthine abscess" with an extradural abscess above the labyrinth.

6. "Angle of the petrosal pneumatic tract," connecting directly with "abscess of the petrous apex." It is only present in an extensively pneumatized mastoid.

7. "Retrofacial osteomyelitic tract," extending inward parallel to the posterior surface of the pyramid (a) above or (b) below the internal auditory meatus and ending in a cavity in the apex.[36] Its external end is separated from the tympanomastoid domain by an area of normal bone.

C. *Petrous Infection Associated With "Lateral Pharyngeal Abscess."*

8. "Lateral pharyngeal abscess of infants," a complication of a "suppurative otitis from osteomyelitic foci in the temporal bone."

9. "Granulomatous osteomyelitis of the apex" with perforation of the abscess into the lateral pharyngeal wall.[37]

V. *Acute Septic Osteomyelitis of the Petrous Apex, Accompanied by a Continuously Positive Blood Culture and Associated With Meningitis Upon the Infection Passing Through the Dura.*

The osteomyelitis may be either (a) hemorrhagic, (b) phlegmonous, or (c) gangrenous in form; (a) "fulminating," (b) subacute, (c) "resting" or (d) "recrudescent" in character; (a) diffuse, (b) invasive, or (c) local in extent; and (a) embolic in origin, or due to (b) an extension of a thrombophlebitis from an adjacent area.

1. Acute fulminating and invasive osteomyelitis: "acute diffuse hematogenous osteomyelitis" of the petrous apex—a bone marrow sinusoidal space infection.

2. Localized hematogenous osteomyelitis.

3. "Subacute thrombotic, (a) localized, or (b) diffuse, osteomyelitis." It may be converted into

4. "A fulminating osteomyelitic phlegmon with an associated phlebitic meningitis," either by operative trauma or upon the advent of a concomitant infection, especially an angina.

VI. *"Embolic Diffuse Hematogenous Osteomyelitis."*

It may be limited to (1) one apex, with an associated (a) embolic mastoiditis and (b) meningeal irritation; or it may involve (2) both apices, because the infection is in the blood sinusoidal space of the marrow.

The embolic character of the infection is early suggested by a (a) spontaneous rupture of the drum membrane; its (b) meningeal character by an (c) intermeatal type of facial weakness, or by (d) pain behind either eye, and the (e) exudate within the pontine cistern, by the absence of, or a great reduction in, the nystagmus induced by turning.

Shortly after invasion of the apex the infection generally becomes (a) semiacute, and then passes into (b) a chronic bone septic stage, or becomes completely (c) resting, resulting in (d) resolution.

However, it may (e) recrudesce, especially if the infected blood channels are traumatized by operation.

VII. *"Recrudescent (Thrombotic) Osteomyelitis Associated With an Immediate and Continuous Blood Stream Infection (Positive Blood Culture) With a Terminal Thrombotic Meningitis or Meningo-encephalitis."*

VIII. *Associated Osteomyelitic Infection of the Marrow-Filled Bones of the Base.*

"Sinusoidal space osteomyelitic phlegmon of the sphenoidal basis," causing diffuse embolic foci, with terminal subdural space meningitis.

IX. *"Chronic Necrosing Osteomyelitis of the Sphenoidal Basis,"*

which, if traumatized by operation, may cause a fulminating meningitis.

THE SURGICAL ANATOMY OF THE PETROUS APEX.

The Petrous Apex, Primordially Filled With Red Bone Marrow, in Relation to Its Neighboring Structures.—The petrous apex, during the growth of the base, is an adaptation of a primordial red-bone marrow-filled proliferating organ, its morphologic pattern being a modification of the spongiose bodies of the vertebræ with sinusoidal hematopoietic spaces; but its manner of growth is that of a growing, proliferating, epiphyseal end of the diaphysis of a long bone.

However, the substantia within the spongiosa of an apex is not dependent for its nutrient upon blood from one artery, as is a diaphysis; for an apex receives nutrient blood not only from the internal carotid artery, but also from the circulatory system of the dura, and from the soft parts of the oropharynx. Thus, because of its specific morphologic structure an apex may be the site of forms of osteomyelitis which resemble osteomyelitic infections of the ends of a long bone or the substance of a vertebra.

The Influence of Infection on a Pneumatized Apex.—When pneumatization extends into an apex, it loses the potentialities which are inherent within its protoplasmic marrow. The protective reaction of a completely pneumatized petrous apex is then carried on by means of leucocytes and other elements brought to it by the general blood circulation, as is the case with the pneumatized bone of the mastoid, the response of a pneumatic apex ceasing to be a local reaction performed by motile histocytes furnished by the red bone

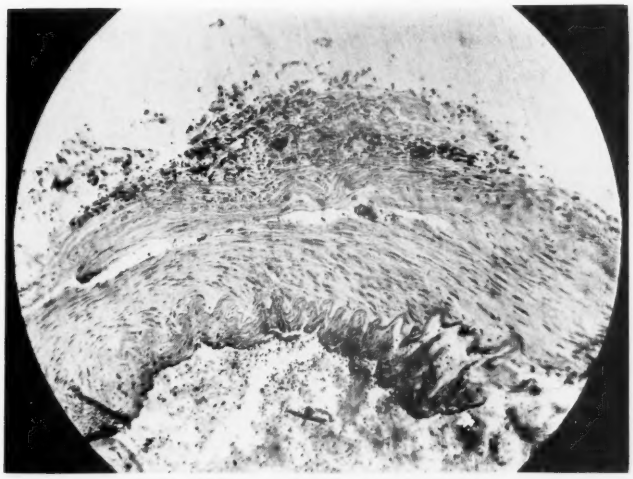

Fig. 1. Section of carotid from the petrous apices. There is periarterial inflammatory cell infiltration but no evidence of thrombophlebitis in the arterial walls which caused an extensive subdural space abscess covering two-thirds of the adjacent cerebral hemisphere.

marrow itself from its own endothelial sinusoidal reticulum, as is the case with red marrow-filled bone.

However, when an apex which is only partially pneumatized becomes the site of infection, its adjacent fat bone marrow, if given time, may control the infection by reverting to its primitive cellular red marrow condition.

Factors Causing the Unique Lesions of Apical Suppuration.—Thus, there are (a) embryologic and (b) growth factors, as well as (c) histologic structural peculiarities of the bones forming the mesial area of the cranial basis that dictate (a) the form a suppurative lesion will assume, and (b) the course that it will take.

Neighboring Vascular Paths of Infection From an Apex.—Of even greater importance is the (c) position of the apex in relation to neighboring vascular structures of the cranial basis and neural tissue. For during the growth of the apex, when the sinusoidal marrow is functioning, its spaces empty into (a) the small plexuses, some of which connect the cavernous sinus with the tympanomastoid area; while (b) the carotid artery and its encircling carotid plexus pass through the apex, and the (c) superior and inferior petrosal sinuses surround it. Any infection of sinusoidal marrow within the bone of

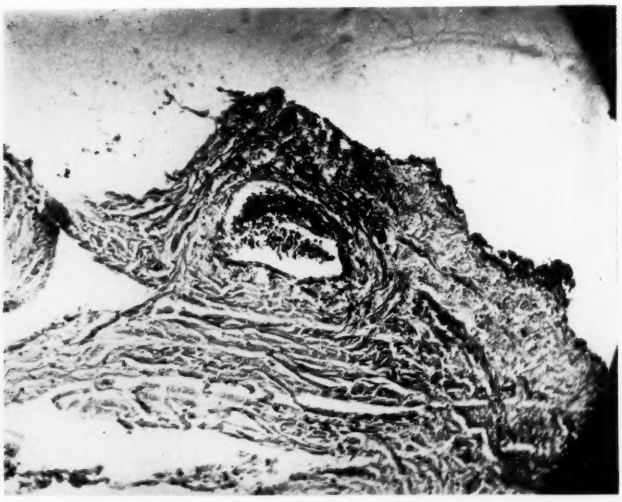

Fig. 2. Thrombosis and inflammatory infiltration in a vessel of the carotid plexus surrounding the carotid artery in its petrous portion, which give rise to phlebitic arachnoid space meningitis.

an apex communicates with the perivascular or vascular channels, and thus infection may be transmitted not only extradurally but also into the subdural or intradural tissue.

An intra-arachnoid infection occurs most frequently, especially while the bone is growing. For the younger the bone, the freer and more intimate are these "receptacular," "rayed" and parvidural[37 38] vascular connections; for most of the smaller plexuses become obliterated at the completion of osseous growth. For there is macroscopic evidence that the petrous apex is the exciting cause of meningitis during infancy in many cases which are usually classified as of unknown origin.

The Early Lesions of Sinusoidal Osteomyelitis of the Apex Are Microscopic Because of the Blood Vessel Nature of the Infection.— In the early stages of all sinusoidal osteomyelitides of growing children the osseous structure appears normal, the only change observable being that the red *bone marrow* has rather a too red or a slightly juicy appearance. However, the normal bone marrow of an apex of a fully grown basis is fatty, and upon the approach of infection this fat may again become red bone marrow, which now is a sign of reaction. At first there are no changes to be seen in it macroscopically.

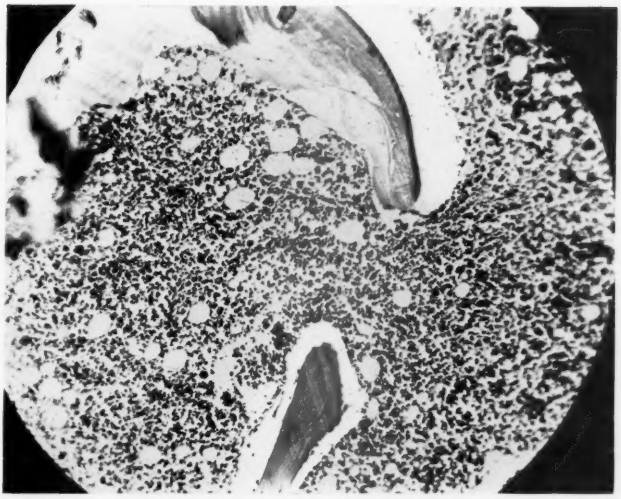

Fig. 3. The sections of the lining of the sphenoid sinus showed purulent inflammation and necrosis. Culture from the left sphenoid: streptococci and staphylococci. Culture from the right subdural abscess: streptococcus mucosus.

Miliary Foci of Suppuration.—Later in the course of the disease there are diffuse miliary abscesses in the marrow, accompanied by radiographic changes in the trabeculæ of the bone.

1. *The Complicating Extradural Abscess or an (2) Associated Phlebitis.*—The miliary foci now coalesce, causing an encapsulated abscess, which may fill the apex, leaving the compact bone of the cortex intact, or may perforate through either the superior or the posterior surface, causing either the well-known (1) extradural abscess over the apex,[34] or a (2) petrosal or cavernous sinus thrombosis.

3. *Phlebitic Subarachnoid Infection and (4) Subdural Space Abscess.*—When, however, a perivascular sinusoidal osteomyelitis of the apex passes out of the bone marrow—as it is primarily a blood space infection—it attacks the coats of the adjacent blood vessels, and thus may cause (a) thrombophlebitis of the carotid plexus or (b) perivasculitis of the wall of the carotid artery. The phlebitis may spread in the subarachnoid spaces as (3) pial vessel meningitis or give rise to a subdural space abscess. (Fig. 3.)

"Erbleichung."—Infection which extends into the cerebral tissues by a vasculitis is evident by a (a) local "Erbleichung" (Spiel-

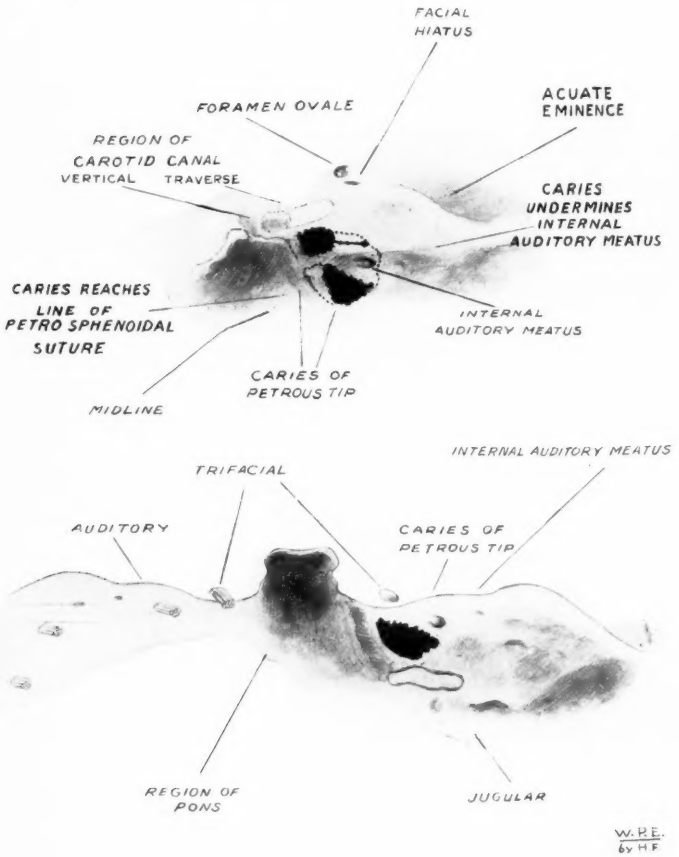

Fig. 4.

meyer[39]) of, or (b) punctate hemorrhages into, the deep cerebral tissues, and is associated with (c) cerebral edema. The macroscopic change in "Erbleichung" is so slight that it is overlooked if not carefully searched for.

SUMMARY.

The lesions of infections of the petrous apex are distinctive: They may be of the nature of (a) a pyogenic sinusoidal osteomyelitis as seen in the sphenoidal basis; or adhere to a type of (b) osteitis so commonly seen in mastoiditis; lastly, infection in a petrous apex may give rise to a unique reactive process within the mastoid, viz., (c)

plasma cell mastoiditis. Because of its embolic origin and specific morphologic structure a pyogenic lesion of a petrous apex may assume one of the forms of (a) sinusoidal osteomyelitis so frequently seen in pyogenic lesions of the sphenoid basis; or if the apex is pneumatized the inflammation may partake of the nature of (b) osteitis or mastoiditis, or be (c) a combination of both. Lastly, infection within an apex may give rise to (d) a secondary specific type of plasma-cell mastoiditis.[31] But in all cases an infective lesion of an apex is (a) pathognomonic and (b) runs a distinctive course, because of the inherent red bone-marrow potentialities of the bone. Consequently petro-apicitis (c) calls for a different surgical viewpoint from that of infections of adjacent osseous tissues, such as mastoiditis, nasal sinusitis or osteomyelitis of the cranial vault, with each of which apical suppuration is a frequent but not an invariable complication, for it may be the primary lesion in the bone.

CONCLUSIONS.

1. Red bone marrow is normally present in the apex during the growth of the skull base, at which time it is a sign of growth. After the early years its presence is apt to be due to the reaction of the bone to infection.

2. The red bone marrow of the sphenoid basis contains a specific biocellular protective mechanism which has a (a) locally immunizing, antibactericidal influence, as well as (b) specific reparative properties inherent within its reticulo-histocytic (macrophage) system. This continues throughout life. A similar mechanism exists in the petrous apex, which contains fat marrow; in which case adjacent air-filled cells may be reconverted into red bone marrow upon the approach of infection.

Consequently, infection within the petrous apex has a tendency to spontaneous subsidence, efficient drainage of the associated mastoid suppuration being all that is necessary to effect a cure.

3. A pathogenic infection of the apex is usually (a) a true osteomyelitis; (b) it runs a distinctive course, and for its successful surgical treatment it (c) requires a viewpoint differing from that of the infective processes so general in mastoiditis.

It is its blood space nature that gives osteomyelitis of the apex its especially dangerous character. The infection within the marrow spreads by way of a phlebitis or periarteritic infection and lights up an obliterating vasculitis of the vessels within the meninges or of the brain substance, particularly in the phlebitic types of infection (pneumococcus and streptococcus hemolyticus).

4. *Symptoms.*—Diffuse osteomyelitic infection is characterized by blood stream invasion—(a) by a chill or chilly feeling. (b) Hyperesthesia or (c) anesthesia of the homolateral cornea is a frequent symptom of apical infection, although it may be present only intermittently. As a corneal abnormality is unknown to the patient, it, together with (d) slight stiffness of the neck, should be repeatedly searched for, especially during the periods when the patient is suffering from a headache.

In the presence of other symptoms of meningeal infection, (e) the absence of vestibular reactions, to turning with preservation of hearing in one or both ears, is of the greatest assistance in the diagnosis of localized pontine cisternal meningitis. The double non-irritability of the vestibular apparatus to stimulation is due to exudate entering the arachnoid prolongation of the internal auditory meatus. (f) There is an "intrameatal type of facial paresis," which is diagnostic of apical involvement. Consequently, in a case of suppurative meningitis if, during the course of the disease there has occurred any pain in the eye, even if seated in the opposite eye, or if there be, or has been, a "suspicion of a facial weakness," the petrous apex should be explored, as it may contain the pyogenic focus which is the immediate cause of the meningitis.

5. Diffuse resting osteomyelitis of the apex may be converted into fulminating sinusoidal space phlegmon by operation.

6. Fulminating phlebitic meningitis is especially apt to be occasioned by an osteomyelitic phlegmon, in which case the blood vessel infection within the meninx is but a part of a diffuse phlebitis involving many vessels in the bone.

7. The operation of "unlocking of the petrous pyramid," associated with a preliminary ligation of the carotid, or a modification of it are the only procedures that fully expose all areas around the ear which may possibly be the "tract" of infection and that allow of adequate exposure of the apex. It should be the method of approach in all patients threatened with or suffering from a complicating intracranial suppuration, as it alone allows of drainage of the pontine cistern mesial to the prolongation of the arachnoid mesh without injury to the brain.

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V. SUMMATION AND CONCLUSIONS OF SYMPOSIUM ON SUPPURATION OF THE PETROUS PYRAMID.*

CXVIII.

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The responsibility for the summation of the data for this symposium has been placed in my hands. It has been accepted with no little misgiving. The president has requested that the emphasis be laid, first, on diagnosis; second, on when operation is indicated; and third, on whether a conservative or a radical operation is indicated.

At first glance, it might seem that such an assignment would be rather simple. However, many difficulties have arisen. I am greatly appreciative of the excellent manner in which the contributors to the symposium have coöperated by sending their papers to me in advance. I would like to have had much more time than I have had to prepare this part of the symposium.

Information gained from the study of statistics, although helpful, seldom can be accurately adapted to the requirements of the individual patient. One needs to be familiar with many facts regarding suppurative disease of the petrous pyramid. In the final analysis, however, the otologist will be required to rely on his own clinical and surgical judgment.

ANATOMIC AND HISTOLOGIC CONSIDERATIONS.

Guild has presented a very comprehensive discourse on the anatomy, histology and surgical relations of the petrous pyramid. He points out that the term, petrous apex, is used by different authors to describe different regions of the pyramid: the surface of the smaller end, the portion anteromedial to the cochlear capsule and the portion anteromedial to the superior semicircular canal. He also brought out that there is so much variation in the size, shape and conformation of the petrous pyramid that a normal can scarcely be described. However, there are certain outstanding features regarding the petrous pyramid that are more or less typical.

The petrous pyramid, according to Guild, may consist of cancellous (spongy) bone with the spaces almost entirely occupied with

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hematopoietic (red) marrow, with an almost pure fatty marrow, or with a mixture of these two types of marrow. It also may consist of a solid dense bone with only a small amount of marrow. Frequently the petrous pyramid is made up of pneumatized spaces, solid bone, cancellous bone with red bone marrow and fatty marrow; there is no regularity to the arrangement of these tissues with reference to each other.

Because the otic capsule is the least variable portion of the petrous pyramid, the position of the pneumatized cells which occupy the petrous pyramid may best be named according to their relation to the otic capsule. For example, those cells that appear below the posterior aspect of the labyrinth may be called the sublabyrinthine cells; those that appear behind the labyrinth may be called the postlabyrinthine cells; those that appear around the labyrinth may be called the perilabyrinthine cells; and those that appear on the anterior surface in front of the otic capsule may be called the antelabyrinthine cells. The antelabyrinthine cells would include the cells around the carotid canal, eustachian tube and those nearer the tip of the pyramid. It is known, of course, that the pneumatization develops from three main points of origin. It starts in the region of the eustachian tube, extends to the middle ear, and then to the region of the tympanic antrum. Cells that originate in the tympanic antrum have been traced by serial section above and around the labyrinthine capsule to the region of the eustachian tube and carotid canal, and vice versa. Cells which originate in the region of the carotid canal and eustachian tube have been traced over the superior aspect of the petrous pyramid toward the tip, or backward behind the labyrinth. The situation of a cell with reference to some near-by landmark is not an infallible indication of its origin. These facts have definite clinical significance, as Kopetzky has stated.

The petrous pyramid is related to very important anatomic structures. Above and behind it is the superior petrosal sinus; below and in front is the carotid artery, the jugular bulb, and the inferior petrosal sinus. Small vessels which arise in the petrous pyramid empty into these various channels. Nothing is known about the lymphatics from this region.

The petrous apex articulates with the body of the occipital bone and with the posterior angle of the greater wing of the sphenoid bone. Occasionally specimens show a marked pneumatization of these regions.

The upper surface of the petrous pyramid is covered with the dura mater of the middle fossa. The posterior surface is covered with

the dura mater of the posterior fossa. The dura in this region is firmly bound down and thinner than the usual dura and may be difficult to elevate from the bone, especially if inflammation is present.

Inferiorly, the petrous pyramid is very near to the lateral wall of the vault of the nasopharynx. This is important because, clinically, cases have been observed in which the suppurative process has localized in the form of a nasopharyngeal abscess.

The trunks, or the roots, of seven cranial nerves, from the fifth to the eleventh inclusive, are in close relation to some part of the petrous pyramid. For this reason, in obtaining complete information regarding disease of the petrous pyramid, one might search for involvement of these various nerves. The jugular foramen syndrome already has been described in connection with suppurative lesions of the temporal bone. The sensory innervation of the petrous pyramid is complex, but the pain which is described as typical of inflammation in this region is said to arise from involvement of the greater superficial petrosal nerve. The tympanic branch of the glossopharyngeal nerve, which sometimes is called the nerve of Jacobson, receives a communicating branch from the facial nerve near the geniculate ganglion; beyond this point it is known as the lesser superficial petrosal nerve and passes through the superolateral part of the perilabyrinth and out along the surface of the antelabyrinthine part of the petrous pyramid, where it occupies a groove very close to the greater superficial petrosal nerve. Through its connection with the otic ganglion it supplies the parotid gland with secretory fibers. Disturbance of parotid secretion may aid in localizing a petrous pyramid infection. The internal carotid sympathetic plexus, which passes through the antelabyrinthine part of the petrous pyramid in the carotid canal, is the direct continuation upward of the cervical sympathetic trunk. All these nerves enter into relation with the branches that are distributed from the sphenopalatine ganglion. Guild suggested that, since the innervation of the dilator muscle of the pupil comes from nerve fibers that course in the carotid sympathetic plexus, differences in the size of the pupils might be caused by an infection near the carotid canal. The dura mater overlying the petrous pyramid is supplied with sensory fibers from the meningeal branch of the ophthalmic division of the trigeminal nerve. The anatomic relation of the petrous pyramid to surrounding structures is very intricate.

CORRELATION OF ROENTGENOLOGIC AND PATHOLOGIC FINDINGS.

The complex and variable anatomic structure of the petrous pyramid makes it difficult technically to bring out, in an x-ray film, important anatomic structures, and also makes it difficult clinically

to interpret any changes in relation to underlying pathologic processes. Clinical experience has demonstrated that comparison of the suspected petrous pyramid with normal petrous pyramids may be helpful but not necessarily diagnostic. Estimation of the general pneumatic development and cellular arrangement in the individual patient may inferentially be helpful and may indeed be misleading. Studies of numbers of petrous pyramids have demonstrated that the petrous pyramid frequently may be pneumatized; whereas, the mastoid process in the same patients may be diploic, and vice versa. If the pathologic condition in the petrous pyramid is osteitis or osteomyelitis, changes in the bone, which are shown by roentgenogram, may occur too late to be of diagnostic value. In pneumatized petrous pyramids one may expect to gain more helpful information. In the present state of our knowledge of the roentgenographic interpretation of changes seen in the petrous pyramids, Taylor has said that "every change in the roentgenographic appearance of the petrous apex does not indicate a suppurative lesion of the petrous apex." Law has said that it is not possible to make a diagnosis of suppuration of the petrous pyramid from the information gained from the roentgenogram alone. It frequently has been suggested that roentgenograms of the pyramid should be made each time a roentgenogram of the mastoid process is made, as such comparative studies should be very helpful in the study of the case at hand, and also in evaluating the clinical significance of future roentgenograms of other patients.

THE CLINICAL PICTURE WITH SPECIAL REFERENCE TO DIFFERENTIATION OF TYPES.

This symposium has considered the actual pathologic changes that take place in infections of the petrous pyramid. Eagleton has classified infectious processes in the petrous pyramid as follows:

1. Reactive and reparative osteitis.

2. Nonsuppurative congestive lesions; symptoms are the result of venous stasis.

3. Chronic sepsis of bone (without microscopic pus).

4. Abscess of the apex.
   (a) Without a tract.
   (b) With a tract.

5. Acute septicemia associated with continuous positive blood cultures and meningitis.

According to the anatomic description, there may be three different types of structure, or a combination of all types and, therefore,

patients whose bone marrow spaces are involved may have an osteomyelitis and those patients whose pneumatized cells are involved may have an encapsulated empyema. Later, an abscess may form or the infection may be of such a nature that it is not limited by suppuration and extends to the intracranial cavity. This may occur in spite of any surgical interference that might be instituted. When it is recalled that the structure is variable, it is conceivable that a combination of abscess of the pneumatized areas and osteomyelitis may coexist. The avenues by which infection extends into the petrous pyramid and causes symptoms are cells which originate around the tympanic antrum, the epitympanic space, the peritubal cells and the cells in the carotid canal. Furthermore, there may be an extensive suppurative process in the sublabyrinthine group of cells without the presence of any symptoms usually assigned to suppuration of the petrous pyramid. It is not an unusual experience to find such an involvement when the mastoid is carefully exenterated according to the suggestion of Küster.

The symptoms of suppuration of the petrous pyramid are pain in, around and behind the eye on the homolateral side; continuous aural discharge during this period of pain, and a low grade sepsis. In addition, a palsy of the abducens nerve may occur. These symptoms may become quiescent and one may be hopeful that the patient is recovering, only to have the symptoms recur. In the terminal stage symptoms of invasion of the intracranial structures occur. Not infrequently one or more symptoms may be lacking and confuse the diagnosis.

The symptom complex of petrous pyramid suppuration may occur in the course of a coalescent mastoiditis or may occur in the convalescent period following a mastoid operation. In the majority of cases that have been reported in the literature this symptom complex has followed a mastoid operation. I would like to interject the thought that this syndrome of suppuration of the petrous pyramid must not be confused with Gradenigo's syndrome. Patients with Gradenigo's syndrome may have pain behind the eye and a palsy of the abducens nerve. This syndrome is not characteristic of any specific lesion.

It would seem that those cases in which an abscess develops would be the only cases in which surgical interference would offer much chance for controlling the disease. If operation is performed too soon in the course of osteomyelitis, before suppuration has taken place or before an abscess has formed, it might offer insurmountable obstacles to successful management. In the treatment of osteomye-

litis elsewhere in the body, surgical interference usually is not attempted until suppuration has occurred.

In the cases reviewed by Seydell, 60 per cent presented quite typical clinical pictures. The onset of the symptoms occurred late in the course of the convalescence following a primary mastoid operation. In his group of cases the palsy of the abducens nerve did not have a definite place in the clinical picture. In the 112 cases reviewed by Eves, seventy-one patients recovered and twenty-nine succumbed as a result of complications. He has added four cases. It can be said that, although there is a certain clinical syndrome associated with suppuration of the petrous pyramid, all symptoms or signs may not be present in any one patient.

THE CHARACTER OF THE PAIN.

The pain is very definite. There is no mistaking the fact that the patient has pain. It is situated in, behind or around the eye. It usually begins at night, and the waking hours may give periods of freedom. The onset of the pain may occur at any time, but usually rather late in the course of the convalescence following a mastoid operation. However, it must be stressed that it may occur before a mastoid operation has been performed. When this occurs it provides definite information which should cause the surgeon to search for evidence of pyramid suppuration. The pain usually is relieved after the suppurative process in the petrous pyramid spontaneously ruptures or is evacuated by surgical effort.

DISCHARGE FROM THE EAR.

After the ordinary mastoid operation one usually expects the ear to be dry within the course of four or five days, particularly if the mastoid has been thoroughly exenterated. If the discharge continues the surgeon suspects that something has been left undone. If, in a given case, the ear has become dry but the patient continues to have pain behind and around the eye, and suddenly the discharge from the ear recurs, these symptoms suggest that a suppurative process in the petrous pyramid has spontaneously evacuated itself, particularly if the pain in the eye has been relieved. If the symptoms persist one may suspect that the drainage is inadequate. If the discharge from the ear has not ceased following a mastoid operation and continues to be profuse in amount or cannot be accounted for on the basis of suppuration in the middle ear itself or in the eustachian tube, particularly if it is combined with the characteristic pain that has been described, one must suspect petrous pyramid suppuration. If the other symptoms of suppuration in the petrous pyramid

are present but there is no discharge from the ear or from the operative wound, then the indication for further operation is more urgent.

THE PERIOD OF LOW GRADE SEPSIS.

Following a mastoid operation or during the course of suppurative otitis media and mastoiditis of the coalescent type, besides a continuous aural discharge and pain, the patient may manifest a low grade type of septic fever. The suspicion that the petrous pyramid is involved in the suppurative lesion is increased. The laboratory aids may not help particularly. The general picture is such that one realizes the patient is not following the usual course for an ordinary suppurative otitis media and mastoiditis, and that some cause must be sought for to explain the sequence of events. The illness is not so severe as one sees in an infection of the blood stream, and none of the signs or symptoms of meningitis are present, other than the headache and fever. The general physical examination does not reveal any evidence of disease elsewhere in the body, and this survery of the general condition makes one believe that the clinical course is caused by disease in the temporal bone. Should the patient gradually recover or the syndrome suddenly disappear, false hope of complete recovery may cause one to overlook the possibility that it is merely a period of quiescence. If the symptom complex recurs, certainly one must consider doing something for the patient. If the terminal stage of intracranial extension supervenes, it is too late. Other findings of importance may be had from an examination of the spinal fluid and the eyegrounds. The roentgenographic examination is extremely important.

In this connection it is very important to stress the importance of eliminating such coexisting infection as suppuration in the sphenoid or posterior ethmoid sinuses. I recall, from my own experience, two fatal cases in the past, which were not associated with the syndrome of petrous pyramid suppuration. Both patients died of meningitis and necropsy revealed that the cause of the meningitis was extension of the infection from the posterior ethmoidal and sphenoidal sinuses. When one realizes that a marked pneumatization of the petrous pyramid and the sphenoid bone may occur, and that infection may spread from either source, one can readily understand how extension to the meninges can easily occur from either source.

FUNDAMENTALS OF THERAPY.

It may be, I think, safely said that the rational method for treatment of such a serious disease as suppuration of the petrous pyramid

is surgical interference. The fact that occasional spontaneous resolution may take place in suppurative lesions of the petrous pyramid does not militate against this point of view. It would seem to me rational to believe that when the diagnosis is well established and the patient fitted into the clinical picture described, that delay in the institution of adequate surgical interference would be detrimental to the welfare of the patient.

In approaching a suppurative lesion of the petrous pyramid, if one accurately has diagnosed the situation of the suppuration, one can proceed directly to this region. In attempting to locate the site of the lesion one may be influenced perhaps by certain features, such as the cellular arrangement, or by the fact that the significance of signs referable to certain cranial nerves may have been interpreted correctly. Still much is to be desired in accurate localization on the strength of physical signs or symptoms. I question, in the present state of our knowledge, whether or not the suppuration can be localized in many cases.

One may well turn back to the dogma of Küster, who recommended that one should be prepared "to open up the bone freely so that it can readily be scrutinized, to clear away all disease and so fully to expose the source of suppuration that the pus is nowhere checked in its outflow." I would grant further that it would be well to keep within the capsule of the pyramid if it were possible. In general, it can be said that one should approach this surgical problem prepared to attack the pyramid in an orderly manner without preconceived ideas or prejudices. One must not speak of conservative or radical surgical interference but should speak of the surgical interference as adequate and rational. Suppuration of the petrous pyramid is a serious disease and heroic measures may be necessary.

If the syndrome of petrous suppuration is present before surgical interference has been instituted on the temporal bone, one may proceed in a purposeful, orderly manner to expose and explore the regions in which the disease may be present. Likewise, if the syndrome appears in the course of convalescence from a previous mastoid operation the surgeon should approach the lesion in the same orderly manner. What I would consider to be an orderly approach would be to explore in turn the cells behind the labyrinth, above the labyrinth, and then to proceed to the zygoma. If careful exploration and exenteration of the cellular elements does not reveal a source of suppuration, or if there are no fistulas to be made out, it then is easy to extend the operation into the so-called radical mastoid exposure to permit the hypotympanum, the region of the eustachian tube and

of the carotid canal to be searched for the source of the trouble. In this latter region one can use the technic suggested by Almour or many use that of Ramadier. Each individual case will be a problem unto itself.

It is suggested that if one is able to keep within the capsule of the petrous pyramid it might be very advantageous, because exposure of the intracranial structures is avoided. However, those of us who have encountered a number of cases of suppuration of the petrous pyramid have found that the lesion may be associated with an epidural abscess, and under these circumstances the intracranial structures have already been exposed by the disease. Further, it is conceivable that, because of the structure and form of the petrous pyramid, one would need to approach the inner aspect by exposing the intracranial structures, that is, by elevating the dura, or perhaps, in order to have adequate exposure, by tapping the ventricle and relieving intracranial pressure so that the brain can be handled easily.

If the lesion in the petrous pyramid is an osteomyelitis, it is difficult to say how the problem should be approached from the surgical standpoint. In the treatment of osteomyelitis elsewhere in the body the surgeons do not approach the lesion unless they feel certain that suppuration has taken place. Waiting for suppuration to take place may result in the onset of the terminal stage of intracranial extension. But could this have been avoided? The nature of the infection is such that it is improbable that the fatal complication could have been avoided. In general, it is evident that there is no one surgical approach that will be adequate or rational in all cases.

CONCLUSIONS.

1. The syndrome of suppuration of the petrous pyramid may be considered to be an established entity.

2. The structure of the petrous pyramid is so variable that the normal can scarcely be defined.

3. Pathologic features of the lesion are fairly well understood. As the pathologic changes are better understood, and the clinical observations more accurately interpreted than they are at present, future studies may be expected to reveal the correlation between the location, the nature of the lesion and the clinical symptoms.

4. The suppurative lesion may be present in the postlabyrinthine, supralabyrinthine, perilabyrinthine, sublabyrinthine or antelabyrinthine regions.

5. The surgical approach to the lesion should be conducted in a purposeful, orderly manner without preconceived ideas or prejudices.

6. Surgical intervention should be described as adequate or rational; not as conservative or radical.

7. The optimal time for surgical interference may be difficult to determine. Each patient will always be a problem for individualized surgical judgment.

CXIX.

VI. DISCUSSION.

DR. S. J. KOPETZKY: I believe that this symposium has definitely established suppuration of the petrosal pyramid as a clinical entity; and the symptoms have been made sufficiently comprehensible so that a diagnosis is possible during life. Dr. Eagleton has given us the philosophy and an outline of the pathology in osteomyelitic lesions. The clinical picture and the course of osteomyelitis, which varies greatly from that presented by osteitis, has not been dwelt upon in much detail. To complete the record I will place this clinical course before you.

According to Ramadier, we deal with two forms of osteomyelitic lesions in the petrosal pyramid. One form is termed hematogenic; and this hematogenic form might very well fit in with the description that Dr. Eagleton gives of his acute septicemic cases. The second form is otogenic. The hematogenic form of osteomyelitic lesion is most frequently found in the very young. The clinical picture is one of general infection. There is marked prostration; there are gastro-enteritic disturbances; and often the signs of pulmonary involvement cloak and cover the advance, step by step, of the infection as it infiltrates the diploic bone, with its contained bone marrow, in the petrosal pyramid. Otitic phenomena are present but the signs and symptoms from the middle ear do not dominate the clinical picture. Eventually, and almost inevitably, the lesion reaches the endocranial structures; and the terminal picture is that of a meningitis. One significant characteristic has been established: the lesion is usually bilateral.

Otogenic osteomyelitis, on the other hand, more clearly takes a course which shows it to be a sequela of otitic infection. This form of osteomyelitis is not confined to infancy or childhood. It occurs,

usually, in the course of a middle ear suppuration. Its onset most often takes place after the initial acute phase of the suppurative otitis assumes a subacute status. Nevertheless, its onset is marked by acute signs. Suddenly, in the course of an otitis which has seemingly subsided into a prolonged otorrhea, there are violent auricular, peri-auricular and hemicranial pains on the side of the head from which there is a middle ear discharge. The tympanic cavity infection simultaneously shows an increased intensity and the discharge from the middle ear becomes more profuse. The temperature ranges from 100 to 102 degrees F. Sleep is disturbed; and often an edema appears over the area of the upper attachment of the auricle. The external canal walls become edematous and painful to touch. This tenderness of the peri-auricular region is in marked contrast to the absence of the usual signs of tenderness of the mastoid process. The painful edematous swelling spreads to the surrounding region, and inflammatory processes become evident in the upper cervical, the occipital and the temporomaxillary region. The complicating lesions which may occur are metastatic, involving the lungs in a bronchopneumonic process, or secondary infectious foci almost anywhere in the body. If allowed to progress uninterruptedly, death ensues from sepsis, pulmonary complications or from meningitis.

Helpful toward furnishing diagnostic data is the use of roentgen films. These show a diploic type of bone structure and locate areas of decalcified bone in the petrosæ, and often in the zygomatic areas. Where evidence of the spread of the lesion is thus presented as being in the squama or in the adjacent bone tissue of the occipital or parietal bone, this diagnosis of the lesion becomes positive roentgenologically. When this evidence is accompanied by a clinical picture as outlined above, diagnosis is established.

With the osteomyelitic lesions, diagnosis is concerned with the recognition of the lesion itself. Where, however, we deal with one or other form of coalescent osteitis, diagnosis concerns itself not only with the recognition of the lesion *per se* but, as Ramadier so well points out in his significant remark, "*It presents itself less as a diagnosis of lesions than as a diagnosis of therapeutic indications.*"

Fundamentally, these diagnoses of therapeutic indications come back to three principles: Recognition of (a) The otomastoidal stage, (b) the intrapetrosal stage, (c) the peripetrosal stage.

The keynote of the various discussions and papers is, in my opinion, found in Guild's, Fowler's, Almour's and Nash's presentations. The otomastoidal stage is stressed by Nash in his discussion

of the so-called chronicities. Almour's nomenclature, and his plea for the study of minutiæ of the clinical picture, fit in well with the hope expressed by Guild, who asks that we take full advantage of all anatomic relationships of the petrous pyramid, both positive and negative, to localize the lesions exactly. Thus we will be able to recognize the intrapetrosal stage and select the proper therapeutic approach which, as Lillie pointed out, must, after a period of competent supervision, be of a surgical nature.

The peripetrous stage, involving veins, with its accompanying phlebitic clinical picture, nerve involvement and dural infections, is also recognizable, and surgical therapy must be prompt to stop the advance of the lesion. The soft palate and the pharynx must be kept under observation to detect gravity abscesses which are also to be construed as of the peripetrous stage of the lesion.

A wide range of difference is apparent between the findings of Guild and the findings of Gordon Wilson, into which I have not time to go. These discrepancies and observations and interpretations need further elucidation and discussion.

Fowler's correlation of roentgenograms with pathology finds us in full accord with him. Like Taylor, he stresses the fact that from roentgenograms alone diagnosis is impossible and should not be attempted. There is one point that was not mentioned, namely, that roentgenograms are the only means by which we can determine the advancement of decalcification. The finding of halisteresis is important. Secondary and subsequent x-rays determine its progress and add diagnostic data. I agree with Dr. Fowler that x-ray interpretation is very difficult, but a trained roentgenologist will always be able to interpret the films.

Dr. Harold G. Tobey: We have listened to a complete exposition of the subject at hand, delivered so clearly and splendidly that there is little to add at this time.

Dr. Kopetzky is to be congratulated on his grasp of the situation and in having gathered up the loose strands of individual experiences and forging them into a clear clinical concept. However, it is inevitable that in emphasizing a particular concept there must be a narrowing of the focus. It is this narrowing that so often makes it difficult for us to fit our individual problems into it.

So fine has been the focus that I have often been confused in the interpretation of the symptoms, and in my contact with others I have found this same confusion.

I have found myself focusing on the closed empyema of the petrous tip to the utter exclusion of involvement of other portions of the petrous and I am sure that others have had the same experience. However, when we recall past experiences with infection of the petrous, with or without sequestration and extradural abscess and the symptoms there presented, the concept has a more familiar ring. Now our grasp of a given subject is inevitably influenced by our personal experiences.

My experience has been that suppuration of the petrous may occur at any level between the base and the tip and give rise to the identical symptoms which have been enumerated today. This may possibly account for some of the differences of opinion as to what constitutes adequate management of a given case.

Let us consider a few of the essential points:

1. All are agreed that such cases occur in the pneumatic type of bone, which of course presupposes a pneumatic type of mastoid.

2. The four periods of progressive involvement are distinct.

(a) Pain.—The typical pain is described as being "retro-orbital, in and around the eye." Now many patients are unable to definitely localize pain, so we may find the pain distributed to the parietal, temporal and mandibular regions also. Pain about the eye and in the temporal regions has for a long time been considered as an indication of dural irritation in the middle fossa. The typical pain may be due to swelling about the ophthalmic nerve in empyema of the tip of the petrous, but we should recognize that less typical pain may mean dural irritation nearer the base of the pyramid or even suppuration of the tip. Here we have an example of the disadvantage incurred by the necessity to emphasize the typical. Kopetzky says, "When the surgical removal of the purulent focus in the mastoid process and middle ear does not result in the cessation of the pain distributed over the areas supplied by the second and third branches of the fifth nerve, the persistence of the pain should be viewed as suspicious of a petrosal tip suppuration"—a statement which may easily be lost sight of when so much emphasis must be placed upon the typical retro-orbital pain.

(b) Aural discharge is almost a sine qua non. I will cite one case in which aural discharge was not present. D. D., a girl of fifteen years, presented all of the typical symptoms and signs, including positive x-ray findings, with the single exception of discharge from the middle ear or mastoid. Under careful and constant observation,

operative interference was not resorted to until the onset of meningeal signs, fortunately with happy results.

(c) Period of low grade sepsis seems to be constant.

(d) I have not observed the period of quiescence.

(e) The terminal stage I have observed all too frequently.

X-ray.—The value of x-ray evidence is none too conclusive and must be considered in conjunction with the clinical evidence. In case of doubt, we should not be satisfied with the evidence presented by the x-ray plate exposed in one position, but should include a basal, an anteroposterior and a Stenver's position. Often the x-ray shows marked involvement of the petrous without clinical symptoms supporting the diagnosis. The petrous may even go on to complete destruction in different areas with ultimate regeneration of the bone without symptoms calling for operative interference.

I believe that if infection of the petrous pyramid is approached with a broadened focus, recognizing that all perilabyrinthine infections, although causing nearly typical symptoms, do not necessarily result in closed empyema of the tip, much of the confusion which is now apparent will disappear.

Dr. George M. Coates: This symposium has been an excellent one and has covered all the points we are ready to discuss at the present time. Petrositis, or petrous suppuration, is not new; the disease has been recognized for years. The fact that the petrous bone is pneumatized and that the spaces may contain suppuration does not mean that radical surgery is always necessary. Before we knew much about the surgery of the petrous bone many of these cases cleared up without surgical interference. I believe we have operated upon these cases at an earlier date than Dr. Eagleton states. I think some confusion has been cleared up as to the distinction between empeyma of the pneumatic cells of the petrous bone and osteomyelitis. The confusion has been partly on account of the fact that we have all types of bone in the petrous; some are pneumatic, some are diploic, some are mixed; there are probably more mixed types than anything else. Most petrous pyramids have some cells in them. I expected to speak on the subject of conservatism in treatment. I think conservatism is needed, but that does not mean that our attitude has to be static. In some cases radical surgery may be real conservatism. In all, I believe there has been an overemphasis on radical surgery. The medical profession at large has got the idea firmly fixed in mind that when a patient shows symptoms of petrous infection there is nothing

to be done but a radical operation on the mastoid and petrous bone. I have been bothered to death by the pediatricians and the internists to perform these radical operations. I have been afraid of the results of this attitude when a case comes into the hands of the younger men. I have seen operations done by some of the younger men which have not been successful, and when operating they did not find the disease. This is not an operation that should be undertaken by a tyro. On the other hand, we have had cases which have died from lack of interference. I had one such case myself. The patient died of osteomyelitis, although there were no symptoms at all until a terminal meningitis supervened. Apparently there was nothing but a mild mastoiditis, healing very satisfactorily. The patient suddenly developed meningitis and died within a few hours. This case had been very carefully watched and there were no symptoms that I could pick up to help me in the diagnosis of the case. We know now, as Dr. Guild has shown us, why the x-rays can be misleading. Key plates should be taken early, which we can use for comparison. Later, when we make subsequent exposures, if they show extension of the disease, we have something definite to go upon. We know also whether we are dealing with the pneumatized type, the diploic type, or the mixed type. We must not forget also that these symptoms may be due to other conditions which will have to be ruled out. These are the cases that have trigeminal pain and those with suppuration of the middle ear with low fever.

DR. HARRIS H. VAIL: My contribution to this symposium is to bring up the importance of the greater superficial petrosal nerve. At the 1932 meeting of this society I asked if the ocular pains so typical in cases of petrous apex suppuration were due to an irritation of the ophthalmic division of the fifth nerve and stated that I felt that these pains were not due to irritation of the ophthalmic division of the fifth nerve but were the result of an irritation of the greater superficial petrosal nerve. (Vail, H. H.: Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous, Apex, Archives of Otolaryngology, February, 1933, Vol. 17, pp. 212-221.) At that time I presented the anatomic relations of the greater superficial nerve, and at the risk of repetition I would like to show you some lantern slides, one from Eckert-Möbius' work and another made from a photomicrograph given me by Dr. Guild. From these cross sections of the petrous bone the close relationship between pneumatic cells in the apex and the greater superficial petrosal nerve is well shown. The eye and orbital pain is very characteristic of petrosal suppuration in its early stage, and inasmuch as great

stress has been placed upon it today it would seem to me highly desirable to know what produces it. No pathologic studies of the greater superficial petrosal nerve have been recorded in the literature because I imagine the nerve was not included in any sections. Several slides taken from Dr. Ziegelman's article also show the nerve in its relations to the petrous apex. It should not be difficult in a fatal case of petrous apex suppuration to make serial sections to show the relations of the greater superficial petrosal nerve, and the findings in such work would answer this important question. It is well known that the greater superficial petrosal nerve carries sensory fibers from the sphenopalatine ganglion to the geniculate ganglion. Ziegelman proposed an operation to cut the nerve in certain conditions which were not well defined by him. I have had the same thought in efforts to relieve the neuralgia due to chronic sphenoiditis. Anatomic investigations have shown that there is a sufficient variation of the point of the nerve's emergence on the front wall of the petrous bone to suggest that it would require at times a much deeper exposure to expose the nerve. Furthermore, it is so firmly bound to the dura that it can easily be torn by elevation of the latter.

Recently I have had the opportunity of seeing a patient who underwent a section of the sensory root of the gasserian ganglion for trigeminal neuralgia and who developed, a few months after the operation, a severe neuralgic pain in and back of the eye on the side of the operation, which pain was not there before the operation. Pins could be stuck in the skin without the patient perceiving them, and yet there was a severe constant neuralgic pain with at times paroxysms when pressure was made on the eyelid or eyeball. All or most of you are familiar with the approach used by the neurologic surgeons for operations upon the gasserian ganglion, and perhaps I need not remind you that after an elevation of the dura over the petrous bone to where the third division of the trigeminal is seen the dura is then incised and the ganglion exposed. In such an operation the greater superficial petrosal nerve can be injured. That such trauma does occur is proven by the complication of a facial paralysis due, no doubt, to hemorrhage or trauma, eventually involving the geniculate ganglion. Dr. Alfred W. Adson, in a personal communication, wrote that he would estimate roughly that about 5 per cent of trigeminal neuralgia cases have burning pains in the face and eyeball after a division of the sensory root. Dr. Harvey Cushing, in a personal communication, states: "In the old days there was a great deal of unnecessary tugging and pulling of the membranes which might easily have injured the superficial petrosal nerve, and whether

this is something which has been avoided in later years with more perfected approach I can't tell."

That this ocular and orbital pain is not present in all cases of petrositis is well known. This fact can be easily explained by a consideration of the route of extension of the infection to the petrous apex. If the infection points towards the posterior surface of the petrous bone of course there is no relationship with the greater superficial petrosal nerve, and there will of course be no ocular pain. However, in such cases the abducens nerve in Dorello's canal is menaced. Therefore I would say that the presence or absence of referred pain in the eyeball or orbit is a very valuable aid in indicating the particular surgical approach.

Dr. F. L. Lederer: A hitherto unknown, or one might say, unrecognized, clinical entity has sprung into prominence by the recognition of a definite symptom complex due to involvement of the petrous apex. The question is whether or not we have established a pathologic entity. Considering only the past year's contributions to the otologic literature, the identity of this disease, which has probably always existed, but which was never so clearly set forth as today, has been firmly established. The contributions in the main are based upon anatomic investigations which render more comprehensive the pathogenesis and therapy of this disease, apparently known to clinicians for the past 73 years.

But on the one hand, the literature presents confusing reports of conservatism, such as expectant management, x-ray treatment, and thorough simple mastoid operations resulting in cures; while on the other hand, there is presented for consideration a more radical surgical attack. A close study of the clinical material reveals that petrositis requires, fundamentally and foremost, a definition simply based on anatomico-pathologic concepts. By such universal principles, as have been presented by the participants in this symposium, types of cases and their particular response to therapy undoubtedly may be better evaluated.

The misunderstanding that prevails among the many contributors to this subject arises from the misinterpretation of findings that unquestionably belong to two entirely unrelated pathologic entities. Suddenly enormous numbers of cases purported to be petrositis spring into being. This very fact would indicate that the term needs clarification. Have we at least a term which would define more clearly the pathologic conditions in those cases that respond to conservatism, which seem to clear up the vast majority of the cases of "petrositis,"

so-called? A conservative attitude is always admirable to hold in check those tendencies that lead toward radicalism, provided, of course, that it is well formulated, but equally discouraging is the attitude which renders, in the course of the disease, even radical measures futile.

As I have said so many times in the past with reference to other problems, we should not make the mistake of formulating hard and fast rules to guide a problem which requires individualism. Only harm has resulted from adherence to dogmaticism. Waves of enthusiasm about clinical entities which are brought before us have resulted in a morbidity and mortality that in retrospect might have been avoided had we studied indications more individually. While there is bound to be an abuse of principles, the misuse of them, at least, may be circumvented by emphasizing individualization rather than generalization. There is no stereotyped manner of judging clinically such involvements and therefore the attack likewise need not be so estimated. Furthermore, there is an inherent danger in dividing the pathologic concept of what appears to be purely a clinical entity.

From clinical experience I have felt that a pneumatic apex—subject to the same possibilities of hyperemia, coalescent infection, empyema and osteitis that may involve the cellular areas of the mastoid and influenced by the existing organism and type of mastoiditis—had to be present to result in the clinical course of the disease which has been so ably described by Kopetzky and Almour. My experience bears out the contention that petrositis, as it is defined by Kopetzky, is not synonymous with the classical Gradenigo syndrome. There is no doubt that a confusion between these two has led to the association of conservatism in the conduct of a case complicated by an abducens paralysis, with conservatism in the management of suppuration of petrosal pneumatic cells in which an abducens involvement is of rare occurrence.

It seems that a beginning in the process of clarifying these conditions might be made by using terms which should be not only descriptive anatomically and pathologically but which also be adaptable for clinical application. I would suggest that the term "*pneumatic petrositis*" be limited to the type that is characterized clinically by deep orbital pain and continued otorrhea. These symptoms, when occurring in the course of a pneumatic petrositis, namely, in the stage of hyperemia of the mucosa of the petrosal pneumatic cells, has been termed by Sunde as *petrosism*, and when occurring later in the osteitic stage has been termed *latent apicitis* by Sjöberg, or "*true petrositis*" by Kopetzky and Almour. The latter type is

approachable by way of the middle ear. As a matter of fact, it is only because there exists in this type of petrositis a cellular development that a separation results, large enough to permit exploration between the cochlea, the tubal orifice, the carotid canal and the tegmen petrosi. The hyperemic stages termed *abortive apicitis* by Sjöberg are amenable to conservatism just as are the hyperemic stages of classic mastoiditis. While the difference may be one of degree, it is nevertheless practical to assume that the subacute and chronic types present the less formidable problem. Further, the most successful of the surgical cases described under this entity have been those in which sufficient time has elapsed to render possible extensive pathologic changes, such as granulation and fistula formation, in the region of the tympanum. This is fortunately the situation which has existed in the greatest number of cases reported.

The other type, which apparently has led to confusion and controversy, I would term "*diploic petrositis*" because it is unapproachable by any other means than by taking down the buttresses as provided for by the Eagleton technic. It is quite obvious that in this type of acellular development the aforementioned surgical entrance of the tip is not available.

The symptoms depend upon the location of the involvement, the degree and extent of same, whether it be by vascular channels or by continuity of structure. The clinical picture of orbital pain, low grade temperature, drowsiness, possible vomiting and nystagmus, excluding the presence of an abducens paralysis and perhaps a swelling of the lower eyelid, seems to be generally accepted. X-ray studies have been made of the petrous apex for many years and, despite the carefully described technic, it is not to be overlooked that a special roentgenologic technic is essential to bring out the requisite details. It has been shown in addition that radiographic evidence alone is not sufficient as an indication for surgical intervention, as it has its limitations and certainly should only be accepted as corroborative. There may be a warning sounded, as has been done by some, to the effect that the rationale of surgical intervention centers in an exploratory procedure without any preconceived method of approach, and in abandoning the tendency toward a mathematical rule in disregard of anatomic and structural anomalies which may exist.

Dr. Fletcher D. Woodward: I wish to briefly outline a few of our experiences in the study and treatment of petrositis and other conditions which simulate it.

1. Pain, when distributed over the first division of the fifth nerve, has been the most suggestive early symptom, and is quite

typical in character. The routine survey of the nose, sinuses, nasopharynx, pharynx and teeth in these cases often discloses pathologic conditions, but as a rule the associated pain is atypical in character, and does not lead to confusion.

2. Otorrhea, when persistent, recurrent or of sudden increase in amount, has been the next most suggestive finding.

3. Abducens paralysis has been found in so many other pathologic conditions that its presence has not been of great significance.

4. Roentgenography has been disappointing in many instances, in that the films were negative in some proven cases of petrositis and positive in others which did not have petrositis. However, I feel that this is largely a question of experience and technic and should be soon settled. In order to bring this about we have made it a routine to radiograph the petrous tips in all cases referred for mastoid examination.

The value of this radiograph may be of great importance, if at any time during the progress of the disease symptoms of petrositis arise, for then we can draw definite conclusions by comparing the first radiograph with subsequent ones.

5. Primary Suspected Petrositis.—Suspected petrositis has been added to the list of indications for mastoid operations, and this is especially true when marked pneumatization is present. When evidence of irritation of the dura is found at operation either by necrosis of plate or epidural abscess, we are content, for experience has shown that dural irritation can produce the typical pain of petrositis, or, if pus is liberated from cells deep within the mastoid and adjacent to the outer portion of the petrous pyramid, we are again content to wait, and watch the progress of our patient.

6. Secondary Suspected Petrositis.—We feel that those cases which have had a previous operation and then present symptoms of petrositis are more apt to have a true involvement. However, in several instances the finding of sequestra, unopened cells, or dural irritation, has been sufficient to clear up all symptoms.

7. True Petrositis.—When none of the above briefly enumerated conditions are found, we are face to face with a most serious problem, one that needs the combined effort of all of us, so that we can eventually place it on the same high plane that mastoid surgery occupies today.

We know from experience that a certain percentage of these cases will drain spontaneously through the middle ear or mastoid and

eventually heal. But since we have no assurance of this happy outcome, it is our duty to devise adequate surgical drainage. Unfortunately there is no well charted course to follow. In our experience the following outline has been evolved:

1. Careful and extensive search for fistulæ, either from the mastoid or from the tympanum in radical operations, offers the easiest and most logical solution, and when found, we are content to watch the progress of the patient to see if adequate drainage has been established.

There are unfortunately certain cases in which careful search of the mastoid does not reveal a fistula, and the condition of the middle ear does not warrant a radical operation. This constitutes one of the major problems to be solved, and in which we have yet no suggestions to offer.

2. When we are unable to discover a fistula either from the mastoid in simple operations, or from the tympanum in radical operations, or when we are faced with the problem of doing a radical when conditions in the middle ear do not justify it, each surgeon must decide which type of surgical approach he will follow, the tympanic approach of Kopetzky-Almour or one of the extradural approaches advocated by Eagleton, Myerson and others.

Our experiences in the surgical drainage of these cases have not been happy, although Dr. Kopetzky was kind enough to do this operation for us on our first case, with an excellent result. I have not had the courage to try it, because of the variability of the angle of approach. In the dried specimens on which we attempted it, the cochlea or the internal carotid canal was too frequently opened. However, I freely admit that the fault may lie in our poor technic, for when it can be done successfully it certainly offers adequate and safe drainage.

My inclinations have been toward the extradural approach of Myerson, or by the old Gasserian ganglion route. In either case, a careful technic must be worked out on the cadaver, with due consideration for the middle meningeal artery, petrosal sinuses and other important structures in this most inaccessible location.

Finally, in those cases in which rupture has taken place and meningitis is either present or imminent, I again feel that an extradural approach offers a better prognosis, and we should bend our energies to the development of a proper technic for surgical drainage in this manner.

The Scientific Papers of the American Bronchoscopic Society.

CXX.

GASTROSCOPY WITH THE FLEXIBLE GASTROSCOPE.*

CHEVALIER L. JACKSON, M. D.,

PHILADELPHIA.

Gastroscopy is attracting widespread interest at the present time, largely because of the development of the Schindler flexible gastroscope. This instrument was devised by Rudolf Schindler,[1] with the help of Georg Wolf, in 1932. It has two great advantages over its predecessors: first, it can be passed almost as easily as an Ewald tube, and second, it affords a good visualization of the antrum and pylorus in the great majority of cases. Benedict[2] reported his experiences with this gastroscope at the Massachusetts General Hospital in March, 1934, and in a second article,[3] which appeared quite recently, he called particular attention to its value in diagnosing and differentiating the various forms of gastritis. Arafa[4] of Cairo, Egypt, has also reported some interesting observations.

In a paper before the Section of Gastroenterology of the American Medical Association, in June, 1934, and since published in the *Journal*, Chevalier Jackson, Sr., and the writer[5] mentioned their first experiences with the Schindler instrument, contrasting its indications and contraindications with those of the rigid open tube. We have now been using the flexible instrument for about a year and a half, during which time we have done more than 100 flexible-tube gastroscopies on over seventy patients. Dr. William A. Swalm, chief of the Gastrointestinal Clinic at the Temple University Hospital, has co-operated with us in this work, and I am preparing, in collaboration with him, a report on the series of cases studied, which will show a correlation of gastroscopic with clinical and laboratory data.

THE INSTRUMENT.

The Wolf-Schindler flexible gastroscope (Fig. 1) consists of a proximal rigid portion and a distal flexible and somewhat elastic por-

*Read before the eighteenth annual meeting of the American Bronchoscopic Society, Toronto, June 1, 1935.

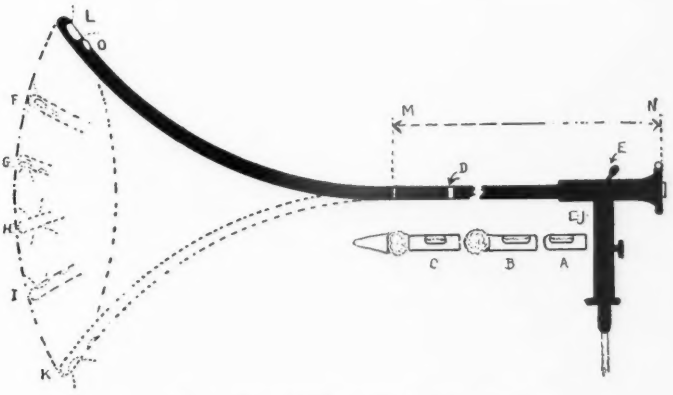

Fig. 1. The Schindler flexible gastroscope.

tion. The essential feature of the instrument is a series of lenses which are placed in the flexible portion in such a way as to permit seeing "around the corner" when the instrument is bent. Several different models have been brought out, some with straight tips, some with bent tips. The instrument that the writer has been using for the past year and a half is one of the bent-tip models, which contains twenty-two lenses and a retrograde optic having a visual angle of 60 . The Schindler gastroscope uses a 6 to 8 volt lamp, which the writer has found most satisfactorily illuminated by means of a dry battery containing six ordinary cells connected in series, with a voltmeter built into the box showing just what voltage is being used (Fig. 2).

TECHNIC.

The preparation of a patient for gastroscopy consists simply in having him take nothing by mouth after the evening meal, and evacuating whatever secretions or retained food there may be in the stomach immediately before the examination, by the simple passage of an Ewald tube. To facilitate complete evacuation the patient should be recumbent on a carriage, the foot of which may be elevated and supported on a box. Schindler uses a special table which permits the patient to assume a modified Sims position for evacuation of the stomach. In any event, it is much better to use simple evacuation rather than lavage in preparation for gastroscopy.

A preliminary hypodermic of morphin and atropin is given about a half hour before evacuation of the stomach, and the writer has found simply spraying the pharynx once or twice with 10 per

Fig. 2. Battery box with built-in voltmeter specially designed for use in connection with the flexible gastroscope. The box contains six ordinary dry cells connected in series. By means of rheostat and voltmeter a constant current of eight volts is assured.

cent cocain a satisfactory method of obtaining sufficient local anesthesia. The spray is used before and after the passage of the stomach tube.

The position of the patient for diagnostic gastroscopy (Fig. 3) is very important. Certainly, regardless of what position is used for introduction, the best position for examination of the antrum and pylorus is the left lateral decubitus. The patient's left elbow should be just back of his body, so that he lies on his left forearm, and the right arm should be allowed to hang over the edge of the table. The head is supported in the head-holding assistant's left hand. As in all other endoscopic procedures, complete relaxation is very desirable.

When the patient has been placed in the correct position, the tip of the gastroscope is grasped in the right hand and introduced into the patient's mouth, using the left index finger to make pressure against the posterior pharyngeal wall and thus prevent the instrument from entering the lower (left) pyriform sinus. The proximal end of the instrument is supported by the nurse during this first stage of introduction. As soon as the tip of the instrument has entered the cervical esophagus the endoscopist quickly advances it down the esophagus and into the stomach. Occasionally slight delay is encountered at the diaphragm. In this event, great care must of course be observed, and only the very gentlest pressure used.

Orientation in the stomach is the chief difficulty, but with practice it can be quickly learned. The simplest method is to advance the gastroscope at once as deeply as it will go, and look for the most

Fig. 3. Position for gastroscopy with the flexible gastroscope.

conspicuous landmark, the plica angularis, which is seen with the white dot on the rim of the ocular at 10 or 11 o'clock. Just beyond this fold one sees the wall of the greater curvature of the antrum, along with peristaltic waves will be seen passing successively toward the pylorus. In most cases the pylorus itself will appear, opening and closing rhythmically. When it is closed or almost closed it has a stellate form (see plate I—1). The other parts of the stomach are examined as the scope is withdrawn and rotated in such a way as to show the posterior wall, the anterior wall and the greater and lesser curvatures in turn. Throughout the examination just enough air is insufflated to secure the desired degree of inflation. Some patients retain the air very well, but others expel it by belching almost as fast as it is introduced.

As a variation in the technic of introduction, the writer has found it slightly easier in some patients to introduce the gastroscope with the patient in dorsal recumbency and the head held in the same position as for the introduction of the esophagoscope by the Jackson "high-low" technic. This position has proved especially useful in sthenic or apprehensive individuals. One advantage of this technic is that the endoscopist can hold the instrument himself from the beginning and keep it perfectly aligned. It must be admitted that there is some objection to using a position for introduction which must be modified after the tube is in place, but as a matter

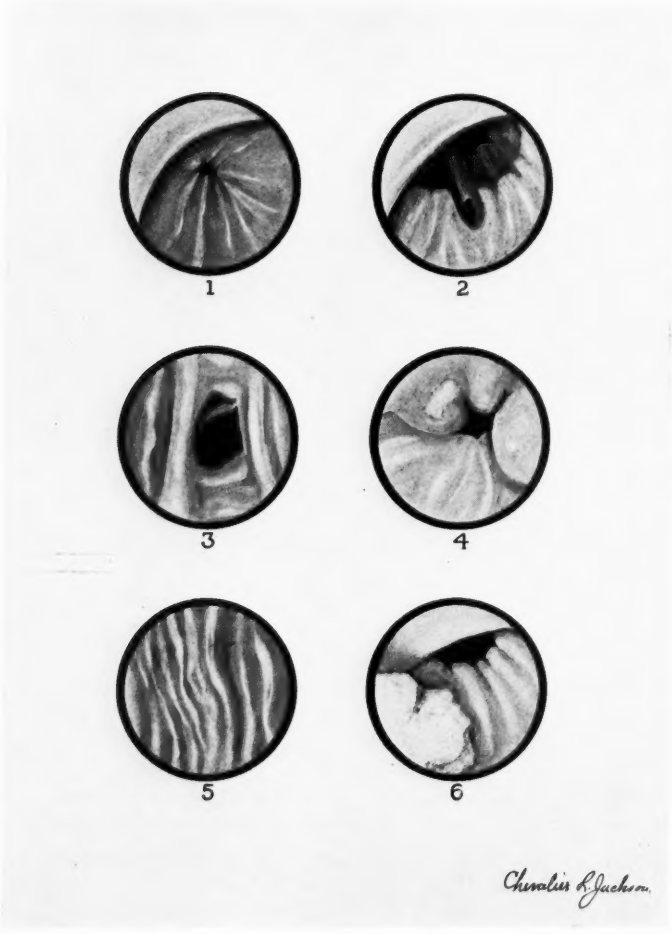

Plate I. Endoscopic views of stomach with the flexible gastroscope.

1. Plica angularis and closed pylorus.
2. Gastrostomic tube as seen in the flexible gastroscope. Note that tube has been placed to the pyloric side of the plica angularis. (After the tube was removed, a special plastic procedure was required because pyloric obstruction developed.)
3. Stoma in a patient with a gastroenterostomy. No ulceration but intense gastritis, probably due to regurgitation because of too great patency of the stoma.
4. Carcinoma invading stomach from outside. (Confirmed at operation.)
5. Superficial gastritis. Note intense congestion and superficial erosion with hemorrhage.
6. Carcinoma involving the anterior wall of the body of the stomach. Partial gastrectomy was done in this case by Dr. W. Wayne Babcock and the operative findings fully confirmed the gastroscopic findings.

of fact the change of position is not difficult if the patient has been instructed in advance.

INDICATIONS AND CONTRAINDICATIONS.

The present renaissance of gastroscopy fulfills the prophecy of Chevalier Jackson,[6] who said in a paper read before the New York Academy of Medicine in 1907, "Gastroscopy is not simply a feat. It has a field of usefulness which will increase as our skill and knowledge increase."

The development of the Schindler instrument has greatly widened the indications for gastroscopy. There is now no reason why the endoscopic examination of the stomach should not become as usual a part of the routine gastroenterologic study of a patient as proctoscopy already is. Not only for the inspection of ulcers or tumors (plate I—4 and 6) already known to exist because visualized by roentgen ray examination, but even more for the study of mucosal changes in the various forms of gastritis (plate I—5) and their better correlation with the other findings, gastroscopy promises to be of the greatest service. Postoperative stomachs (plate I—3) may often be studied to better advantage by gastroscopy than by x-ray. Almost any complaint referable to the gastrointestinal tract constitutes an indication for gastroscopy, provided careful preliminary study has not shown any contraindication to exist.

Contraindications to the use of the flexible gastroscope consist of: stenotic or ulcerative lesions of the esophagus, aortic aneurysm, esophageal varices, or any lesion of the cardiac portion of the stomach involving the lower end of the esophagus. These lesions, with the exception of aortic aneurysm, would not contraindicate careful examination with the open-tube esophagoscope or gastroscope; in fact, they would constitute indications rather than contraindications.

I have seen many patients with disease of the lower end of the esophagus simulating disease of the stomach, and I have been impressed with the importance of careful fluoroscopic and roentgen ray examination of this area, followed, in many cases, by careful open-tube examination, even though the patient's complaint may have at first seemed purely gastric. A surprisingly large number of patients, first thought to be suffering from purely gastric disorders, have been found to have ulceration of the lower esophagus associated with "short esophagus" or hiatal hernia; others have had carcinoma at the esophago-gastric junction. In one case, carcinoma of the stomach was diagnosed by retrograde open-tube gastroscope with biopsy in a patient who had also a stomach partially above the

diaphragm. Among the indications for the use of the open tube are: the taking of specimens for biopsy, the local treatment of benign lesions, and the removal of foreign bodies from the stomach.

Granting that it has its contraindications, and that for certain purposes the open tube is better, there is no question that the development of the Schindler gastroscope marks a new era in the endoscopic study of the stomach, and the day is not far distant when every good gastro-enterologist will make gastroscopy a part of the routine study of his patient. For us, as specialists in peroral endoscopy, the flexible gastroscope, while a valuable addition, will not entirely supplant the rigid open tube.

3432 N. Broad Street.

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CXXI.

A PRELIMINARY SURVEY OF THE EFFECT WHICH LYE LEGISLATION HAS HAD ON THE INCIDENCE OF ESOPHAGEAL STRICTURE.*

H. MARSHALL TAYLOR, M. D.,

JACKSONVILLE, FLA.

Every member of the Bronchoscopic Society is doubtless familiar with the fact that the Federal law providing for the proper labeling of packages of household lye and other caustics was passed by Congress and signed by the President of the United States in 1927. It is doubtful whether this law would ever have been enacted but for the ceaseless and untiring effort of Dr. Chevalier Jackson. Dr. Jackson not only sensed the importance of this legislation but through sheer force had it passed in his State of Pennsylvania and then was responsible for it becoming a Federal law. Dr. Chamberlain has well said that Dr. Jackson's attitude in this cause has been another evidence of the altruistic spirit which distinguishes and glorifies medicine.

At the midwinter meeting of the Council of this Society, Dr. Forbes, our President, requested that a report be made at this session relative to this medicolegal matter.

A preliminary survey indicates that the lye legislation enacted as the result of the work of the Committee on Lye Legislation of the Section on Laryngology, Otology and Rhinology of the American Medical Association is beginning to bear good fruit. Complete and fully representative statistics cannot be presented at the present time, but the data so far obtained are valuable as an index of what can be done.

In Pennsylvania, which was the first State to enact this law, there has been observed a decrease of more than 50 per cent in the number of lye burns since this legislation went into effect. Statistics accumulated by Dr. Jackson showed 102 cases of lye burns in one year preceding this law, while in 1934 there were 53 cases. In Florida, which was the second State to enact this law, in the year preceding this enactment fourteen cases of lye burns were reported, while in

*Read before the eighteenth annual meeting of the American Bronchoscopic Society, Toronto, June 1, 1935.

1934 there were but six cases. Dr. Carmody, in a communication to the writer, advises that there has been a reduction of more than 75 per cent in the number of cases of lye burns in the State of Colorado since this preventive legislation went into effect. If these three states, each representing different sections of the country, can be used as an index, this committee, headed by its distinguished chairman, Dr. Jackson, should feel that its efforts have been more than repaid.

In evaluating the effect of this legislation there is another important consideration, namely, that the use of lye has enormously increased during the interval. A great many new caustic preparations have been added in the form of drain pipe cleaners, paint removers, etc. Very extensive advertising has also increased the use of these caustic preparations. For this reason it would seem that we are justified in assuming that there is at least twice as much lye used now as compared to eighteen years ago.

I feel sure that I voice the sentiments of Dr. Jackson when I say that the members of this Society will be recreant to their duty if they allow their interest in this subject to wane. It is most important that the different phases of it should be borne in mind from both the humane and economic standpoints.

111 West Adams St.

CXXII.

BRONCHOSCOPY IN A PULMONARY DERMAL INCLUSION CYST.*

JOSEPH C. BECK, M. D.,

CHICAGO.

The opportunity for making bronchoscopic observations in a case of a pulmonary dermal inclusion cyst is so infrequent that the incident of such a case was thought to be of sufficient interest to be reported.

In all probability inclusion cysts of the pleural cavity of the chest wall or the lungs will become increasingly more rare. Their greater incidence several decades ago was due, in a large measure, to the open method of treating chronic empyema and chronic pulmonary suppuration by means of various plastic procedures for obliterating the suppurative cavities. It not infrequently occurred during the plastic inlay that dermal tissue was permitted to become buried, thus establishing a nidus from which a small cavity filled with epithelium could grow, forming a pocket not unlike that of a cholesteatomatous sac.

The change in technic of surgery in chest conditions, especially the replacement of the open treatment of pulmonary and pleural suppuration by the closed treatment that is in vogue today, will, of course, engender a distinct lessening in the incidence of the use of plastic flaps in this area and subsequently a marked decrease in the appearance of these dermal inclusion cysts.

The following case history will present the salient features of a case that is illustrative of most of those seen.

REPORT OF A CASE.

CASE 1. Mr. P. S., age 27, had been seriously ill from a severe case of pneumonia complicated by a pleurisy with effusion. This was treated by open operation, and the insertion of a drainage tube in the pleural cavity. For some reason a chronic suppuration ensued, and he wore the drainage tube for a period of three years. At this time the patient was afflicted with another attack of pneumonia from which he successfully recovered. However, he continued to expectorate a

*Read before the eighteenth annual meeting of the American Bronchoscopic Society, Toronto, June 1, 1935.

great deal of putrid sputum following his apparent recovery. This persisted for some period of time and he finally presented himself to Dr. Emil G. Beck for treatment in 1917.

The external chest findings were of no interest to a bronchoscopist. However, the sputum examination disclosed, besides pus cells and a large number of various micro-organisms, a good number of elastic tissue fibers. Radiologic examination also disclosed evidences of a pulmonary abscess. He was then operated upon for this pulmonary abscess.

Under general anesthesia a wide thoracotomy was made, including the excision of a number of ribs and also the resection of a large portion of the middle lobe on the right side. The patient had a rather uneventful recovery from this procedure and the wound was subsequently permitted to heal by granulation formation.

Some four months later the usual plastic sliding skin flap operation was performed to obliterate or cover the chest defect. All flaps healed perfectly, except a small fistula which communicated with the bronchus, thus establishing a bronchial fistula. Dr. Beck then proceeded to cauterize the fistula until it finally closed. Some three or four months after the successful closure of the fistula the patient returned complaining of a spasmodic cough, which came on at certain periodic intervals. The sputum that was expectorated was peculiar in that it consisted mainly of masses of foul smelling material rather than the old purulent secretion that he complained of prior to the operation. The expectoration and cough persisted for a day or two and then the patient would remain free of both cough and expectoration for a period of several weeks, only to return with the same complaint. At this time he was referred for bronchoscopy.

X-ray and fluoroscopic examination showed the presence of a possible abscess in the middle lobe adjacent to the chest wall and it was thought that in all probability he had a recurrence of his pulmonary suppuration. Bronchoscopy disclosed that the trachea was somewhat injected. The right main bronchus was filled with a great deal of secretion that consisted mainly of an accumulation of thick whitish material and detritus. Upon suction and cleaning of the right main bronchus it was seen that the debris was coming from the middle lobe. Investigation of this bronchus showed that it terminated in a cavity about four centimeters in diameter. This cavity was filled with a white grumose material similar to cholesteatoma. Upon suction, the cavity was finally cleared of its contents and was seen to be lined with white soggy material not unlike epithelium.

The material removed by suction was then subjected to microscopic examination and was definitely established to be of dermal origin. It was obvious then that we were dealing with a dermal lined cyst. The diagnosis of a dermal inclusion cyst was thus definitely established.

Undoubtedly in the shifting of one of the skin flaps and effecting the closure of the chest wall some dermal tissue had been turned in and permitted to grow in place or else the ingrowth of the skin epithelium that finally joined the mucous membrane of the bronchus in forming the bronchial fistula had been shut off by the use of the cauterization of the external portion of the fistula. The buried epithelium then had grown with the subsequent formation of the epithelial cyst. The question of therapy remained to be solved. It was decided to resect the dermal cyst with the galvano cautery by the aid of the bronchoscope.

Under local anesthesia the bronchoscope was reintroduced into the cyst. Using the transmitted light of the bronchoscope through the chest as a guide, Dr. Emil

G. Beck incised the external chest wall with a pointed galvano cautery until he opened into the cavity. The bronchoscope was then withdrawn. Examination of the exposed cyst showed it to be lined with definite epithelium and was more or less spherical in appearance. The skin lining was then destroyed by means of a large flat galvano cautery electrode and the cavity subsequently packed with iodoform gauze. The cavity was permitted to heal by the formation of granulation tissue until it had been entirely obliterated and then the skin was permitted to close over it. The patient made an uneventful recovery. He returned for periodic observation and was repeatedly bronchoscoped. He was last seen in 1925 by my associate, Dr. M. Reese Guttman, who bronchoscoped him. At that time it was found that the bronchus ended in a blunt sac with no evidence of dermal proliferation.*

185 N. Wabash Ave.

*Photographs of patient and schema of formation of cyst were illustrated by two lantern slides.

CXXIII.

ESOPHAGEAL ACCIDENTS.*

THOMAS E. CARMODY, M. D.,

DENVER.

When we think of our modern methods of diagnosis and examination of the tubes and cavities of the body we have more respect and sympathy for the practitioners of medicine of the last century and before.

The discovery of the roentgen ray in 1895 and the use of the direct laryngoscope and esophagoscope by Killian, Ingals and Coolidge in the early part of this century and the great development by our own Jackson and Mosher since that time have placed us in a much different position.

Those of us who first saw the light of day and spent our childhood on a farm may remember the use of the handle of some barnyard tool to assist the swallowing of a potato by a cow. This is the same as the forcing down of smooth foreign bodies with a stiff tube like the ordinary stomach tube. If the body is smooth and not excessively large no harm will be likely to result, but this was not always known. The former method of passing the esophagoscope with obturator might be as dangerous, and we have most of us been guilty, even though very few cases have been reported. It might be well to report these cases in the plural, which would be a way of dividing the responsibilty as we do in other cases, but I am very much afraid that I must take the blame in the great majority of my cases.

While the case reports are not numerous, the following causes may be noted:

1. Perforation by a foreign body without instrumentation, which is usually due to muscular action or to attempts on the part of the patient to force the foreign body downward with food, water or other means or due to delay, allowing ulceration due to dull bodies or perforation in some way by sharp bodies.

2. Perforation by infection of the esophagus due to ulceration followed probably by straining in cases of typhoid fever, whooping

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cough, or other wasting diseases, in which case we usually find air in the tissues.

3. Perforation due to attempts at removal of foreign bodies blindly by the use of either dull or sharp instruments, stomach tubes, hooks, probangs, etc., for which there is no excuse at present.

4. Injury or perforation by use of the esophagoscope, as formerly used with obturator, or even under the eye, as in cases of carcinoma or those cases where previous instrumentation has damaged the wall, aneurism of the aorta, syphilitic or tuberculous ulceration, or foreign body imbedded in the wall or completely through the same; in the latter case by stretching the wall and enlarging the opening already present. I reported a case last year before this society which may have been of this type.

5. Instrumentation through esophagoscope without the use of the fluoroscope. This may involve injury to the walls by direct perforation, by separation of muscle fibers or scar tissue, by injury to a vessel producing an embolus or by sudden spasm of the esophagus in a nervous patient, producing one of the injuries above noted.

Most of these accidents can be prevented by proper care, careful history taking and good x-ray pictures and very careful instrumentation.

REPORT OF CASES.

CASE 1. Mr. Z. consulted me in May, 1917, with difficulty in swallowing and loss of weight. He was examined with esophagoscope under ether and a stricture was found at the crossing of the aorta, which had already been shown by x-ray. Considerable redness and swelling of the esophagus and slight edema were present, but, while malignancy was suspected, we felt that a bougie could be passed. This was done but without the esophagoscope as we did not have the small flexible bougies.

Immediately after passing the bougie the patient's breathing became labored and he died about ten hours later.

There was very little bleeding, but probably blood filled the mediastinum.

No autopsy was performed.

CASE 2. A. M., girl of 10 years, was seen in consultation with Dr. Richards. She gave a history of ten days' illness with fever, diarrhea and some cough. Typhoid fever was suspected, but within the last few hours swelling of the neck, difficulty in swallowing and breathing had complicated the other symptoms. On examination nothing could be found in the pharynx or larynx but the neck was very much swollen and on palpation of right side of neck and chest marked crepitation was elicited. She was not esophagoscoped and died within twenty-four hours.

CASE 3. S. A., child, a boy of two and a half years, came to Children's Hospital in December, 1923, with a history of injestion of lye solution three weeks before admission.

The patient was unable to swallow solid or semisolid food, and fluids with difficulty.

On December 10, he was esophagoscoped with Mosher esophagoscope and a small Lynah bougie was passed, apparently into the esophageal lumen. This bougie is metallic and not flexible.

Difficult breathing did not appear at once but was noticed as the child was recovering from the anaesthetic. On examination of the chest, the right side showed the presence of air and, later in the day, fluid. The pleural cavity was needled and fluid withdrawn several times in the next four or five days, but no air was reintroduced and the patient died on the sixth day.

Autopsy was not permitted.

CASE 4. I saw Mr. S., age 22 years, at St. Luke's Hospital on June 21, 1925.

Patient gave a history of swallowing a wooden tooth pick about six hours previous and of pain in chest since that time. Barium liquid and capsules gave very little information. Esophagoscopy was performed, but no foreign body was found although a small spot on the left side of the esophagus about the bronchial constriction, which showed some blood, was noted.

He continued to complain of pain in chest and twenty-four hours later, having 102 degrees temperature, we opened the mediastinum and drained with rubber tube drains. This was done by a general surgeon, but the patient died forty-eight hours later.

No autopsy was allowed.

CASE 5. S. D., a girl, three years of age, was referred to Children's Hospital with the following history:

While confined in another hospital for an attack of pneumonia, the child was given a teaspoonful of solution of sodium hydroxide by mistake instead of cough mixture. How this solution came to be in a medicine cabinet could not be explained, although a thorough investigation was made.

After acute symptoms subsided, the patient came to us and an attempt was made to dilate with Jackson flexible bougies.

On first attempt dilation took place easily and patient was able to swallow much easier. Two weeks later the operation was repeated, but the patient had difficulty in breathing as soon as the bougie reached the stomach. She was returned to bed and stimulated and fluids given only by rectum and the symptoms disappeared in three or four days. Fifteen days later the procedure was repeated and the lumen of the esophagus seemed patulous and the bougie passed without difficulty. However, respiration became labored and the child collapsed, and lived only forty-six hours.

Autopsy showed that the bougie passed into the stomach, although there was slight hemorrhage under the mucosa at the cardia. However, a perforation of the esophagus beginning opposite the cricoid cartilage and continuing to the diaphragm but separated from the pleura by connective tissue was filled with partially healed tissue apparently several days old.

Apparently the passing of the bougie fifteen days previous had gone outside the esophagus, but had healed and death was apparently due to embolus or shock of later bouginage.

CASE 6. Mr. A., age 30, came to my office on September 10, 1930, but was in such a condition after traveling 300 miles that he was sent at once to hospital and was seen by me immediately, because of difficult breathing. A tracheotomy was performed without anaesthetic, but the patient died on the table. A great amount of blood was found in the tissues surrounding the esophagus and trachea, but there was no difficulty in getting into the trachea.

A history was obtained from his wife. The evening before, while eating duck, he choked, and after this could swallow only liquid.

He consulted a doctor who used a probang to try and dislodge the foreign body and the patient complained of much more pain and was unable to swallow even liquids. This was about eighteen hours before he came to us.

Autopsy showed the mediastinum filled with blood and there was a duck rib with curve downward, both ends protruding through the esophagus at the level of the tracheal bifurcation.

Why this patient had lived nearly twenty hours is inexplainable.

CASE 7. H. A., male, age 50, consulted me regarding a tooth plate in the esophagus, which had been present forty hours. Three attempts, one with the esophagoscope, had been made at removal. As he was unable to swallow liquids, he was examined with the esophagoscope, but removal was not attempted. After sixty hours of rectal feeding and giving of large amounts of fluid, an external esophagostomy was performed, but patient ceased breathing as the esophagus was exposed, but before opening.

No autopsy was performed, but it was believed that death was due to embolus.

CASE 8. A boy, age 10, had a jackstone in the esophagus and was believed to be in such condition that he could not make the trip from Montana to Denver.

After a phone consultation, external esophagostomy was advised and performed with removal and complete recovery.

Ten months later he swallowed a radish, which lodged at the same spot as the former foreign body. The patient started on his bicycle for his home, which was some little altitude above where he was at the time of lodgment of foreign body and required some extra effort. On reaching home he collapsed and died, and it was found at autopsy that the esophagus had ruptured through the scar of the previous operation.

These accidents, like auto accidents, were mostly due to haste or bad judgment, or both.

Some of these patients could be saved today by the use of mercury tube dilators or Tucker retrograde bougies.

Many near fatalities could be recited in cases where nature was kind.

There is one other case not included here in which the patient refused esophagostomy and I foolishly passed a bougie, and immediately he complained of pain in the region of the diaphragm. He was taken home and continued to suffer for several hours, when a general surgeon was called and he was opened up at Mercy Hospital,

in my presence. No perforation was found; nothing was found to account for the pain. It simply, apparently, was a spasm of the esophagus. The patient died, either through my efforts or those of the general surgeon. We both took the blame.

That ends the cases of fatality, though we have had many more of other kinds

METROPOLITAN BUILDING.

Clinical Notes and New Instruments.

CXXIV.

TREATMENT OF A SERIES OF CASES OF SO-CALLED CARCINOID TUMORS OF THE BRONCHI BY DIATHERMY: A REPORT OF TEN CASES.*

JOHN DEVEREUX KERNAN, M. D.,

NEW YORK.

The so-called carcinoid tumors of the lung, a series of which I am reporting, have only recently been differentiated from true carcinomas. In the Archives of Surgery, January, 1929, Dr. Arthur J. Cracovaner of New York City and the present author reported one of these cases as a carcinoma of the lung cured by diathermy. As more of these tumors have been encountered, it has been possible to check their history and course more carefully, and at the present time it is evident that they form a very distinct class by themselves. It is very important that they should be properly differentiated from true carcinomas, for even now competent pathologists report them as carcinomas, and, on the basis of that diagnosis, a pneumonectomy is proposed. As I hope to show, much less serious surgery is required for their cure.

As a rule, these tumors are found in the larger bronchi, most often in a primary bronchus. They take their origin beneath the surface of the epithelium, in all probability from some of the cells in the walls of the ducts of the glands. They grow freely in the lumen of the bronchus and only after a somewhat prolonged existence have they any tendency to invade the lung tissue. It may be said with certainty that they never metastasize or infiltrate, but, as they increase in size, they compress the surrounding tissue and cause atrophy. Their microscopic appearance is very characteristic. The sections show anastomosing columns of cells which in places have a tendency to form gland-like structures. It is on the basis of this appearance that some pathologists report them as benign adenomata. (Figs. 1 and 2.) The figures which are shown here illustrate the appearance very well: the

*Presented before the meeting of the American Laryngological Society, Toronto, Ont., May 31, 1935.

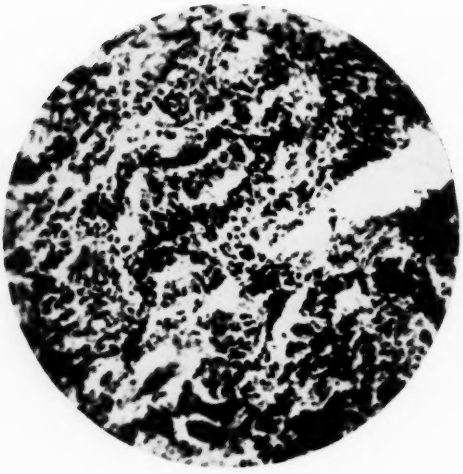

Fig. 1.

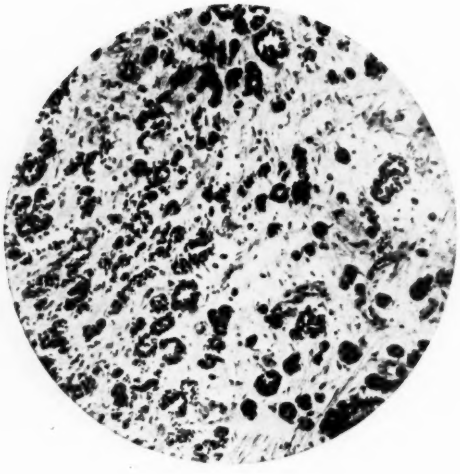

Fig. 2.

first one, the anastomosing strands of cells, and the second the gland-like formation.

As I have said, they grow beneath the epithelium of the bronchi. In none of the microscopic sections has the surface epithelium been found broken through. Fig. 3 illustrates this point. As they continue to grow larger, they eventually break through the bronchial wall and invade the lung tissue, not by infiltration, as has already been said, but by an increase in size which causes a pressure atrophy. Fig. 4 enables you to form an idea of their continuous life history from a small tumor growing into the lumen of the bronchus to a large tumor almost completely replacing two lobes of the lung.

The symptoms are mainly those caused by obstruction to the bronchus. This would mean that when small, they are symptomless, unless, as it occasionally happens, there is bleeding. In four of the ten cases I am reporting this symptom of bleeding was found, and other reporters have reported it even more frequently. Once the tumor is large enough to obstruct the bronchus, serious symptoms may begin. These may be mild: merely cough and more or less sputum. As the area below the obstruction becomes infected, a bronchiectasis results. Then occur severe attacks of coughing, dyspnea, cyanosis and high fever, which are often considered to be bronchopneumonia or lobar pneumonia, according to the physical signs and the x-ray appearance. Often the obstruction may be so severe as to cause a complete massive collapse of one lung with all of the signs accompanying that condition, namely, fixation of one side of the chest, elevation of the diaphragm, pulling over the heart and mediastinum, and severe dyspnea and cyanosis.

The x-ray appearances are most varied. If only a small bronchus is obstructed, a limited shadow of collapse will appear in the x-ray films. If one of the main bronchi is obstructed, all lung markings on one side of the chest will disappear.

The bronchoscopic appearances are very characteristic. The tumors are rather soft, give a suggestion of being pedunculated, and may even move with respiration. The wall of the bronchus is not fixed and indurated, as it is in true carcinomas, so that respiratory motion can be seen except when the tumor is very large. The tumor itself is quite vascular and bleeds easily when touched with the bronchoscopic instruments or when a biopsy is taken.

Once the diagnosis is made, the question of therapeutics urgently presents itself. The treatment proposed by various authors has varied from none at all to pneumonectomy.

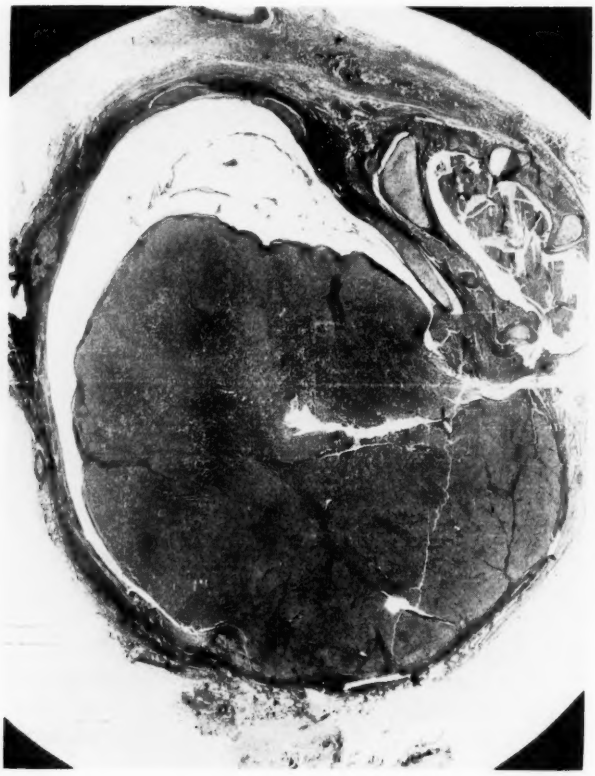

Fig. 3.

As an example of no treatment, I may quote the case of L. K., Case 6. Here was a tumor involving both main bronchi, of sufficient size to deprive the left lung of its function, and on its way to do the same to the right. The prognosis appeared to be exceedingly bad. Several pieces of tissue were taken for examination. The diagnosis at that time was "probably small-celled carminoma." This woman was rather better two days later on discharge from the hospital, the left lung showing more aeration. Nevertheless, it was felt that she was being sent home to die. She came back to the hospital eight months later, seemingly in good health. An x-ray examination of the chest showed clear lungs. No trace of the tumor could be found on bronchoscopic examination. The mere taking of a biopsy appeared to be sufficient to check the growth of this tumor and reverse its course.

Jackson some years ago reported a case in which a small tumor was found in one of the bronchi, probably of a similar nature to these which I am reporting. That tumor was removed by forceps and there was no recurrence.

In one of my cases, H. G., Case 2, a short series of x-ray treatments was sufficient to check the chief symptom, which was hemoptysis. That patient has been followed for eight years and has remained well, although there is still a shadow in the lung, at the site of the tumor.

Taking of biopsies certainly will not cure every case. The first of these tumors encountered by the author, A. B., Case 1, was treated by the implantation of radon seeds and the application of diathermy. Several large portions of the tumor were removed in the making of biopsies. In spite of all of this trauma, it persisted for two years and only thorough coagulation finally destroyed it.

One case of the author, A. J., Case 3, was beyond all help by bronchoscopic treatment when first encountered. In spite of implantation of radon seeds and many attempts at coagulation, the tumor persisted and manifested its presence by extremely profuse hemoptysis. Finally, in a desperate attempt to save her life, a lobectomy was done, to which she succumbed.

It has been necessary in several cases to consider, in addition to the treatment of the tumor itself, treatment of the accompanying bronchiectasis. As a rule, bronchiectasis is always present once the tumor has become large enough seriously to obstruct a large bronchus. Usually removal of the tumor will cure the bronchiectasis, or at least permit it to become dry. In Case 1, after the tumor had been destroyed, all pulmonary symptoms were relieved. There are many cavities in the lung but they are empty and sterile.

In another one of the author's cases, Case 4, in spite of the fact that the tumor was successfully destroyed, bronchiectasis persisted and finally the symptoms became so severe that a lobectomy was called for. This was successfully performed and the woman is now well. It is of interest to note that none of the original growth was found in the lobe removed, which would demonstrate a successful destruction of the tumor. One cause of failure may be a persistence of the stricture of the bronchus after removal of the growth. In author's Case 7, a boy of seventeen, the growth was sufficiently removed, but the work was only too extensively done, as there occurred at the site of the growth a fibrous stricture. Repeated infections recurring beyond this stricture and insufficient drainage

Fig. 4.

because of it finally resulted in such severe invalidism that a lobectomy had to be performed. This was done without success, the patient succumbing to the shock of the operation. Here also, as in Case 4, examination of the specimen showed no trace of the original growth.

With increasing experience and greater perfection of instruments it has been possible to secure quicker and better results in the destruction of these tumors by diathermy. The original applicator used by the author was one improvised from a suction tube through which was led a long piece of tonsil snare wire. It was insulated by drawing over it a piece of rubber tubing. This could be passed through the bronchoscope and the end plunged with more or less accuracy into the growth. Once in place, the wire was touched with an electrode leading from the diathermy machine, thus completing the circuit. You will realize at once that the bulk of this improvised instrument prevented any accurate placement of the wire, and, moreover, prevented observation of the effects of the current on the growth. It was necessary to guess when sufficient current had been applied, and often its removal and replacement several times during the course of one treatment was called for. Inaccurate placement was also a

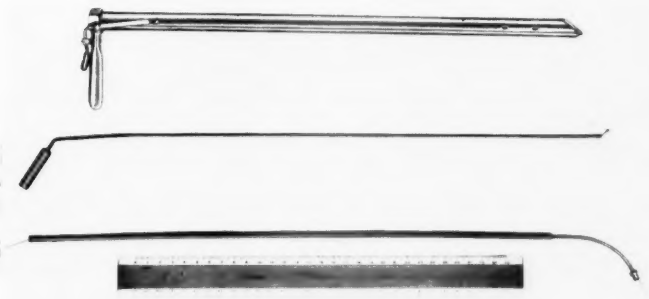

Fig. 5.

danger and I think, accounted for the stricture which occurred in Case 7, already described above.

The perfected instrument (Fig. 5) allows most accurate placement of the tip and observation during the whole course of application of the current. The degree of the destruction of the growth may be seen and the current stopped once the operator judges the procedure has been carried as far as is safe. You will note that the tip takes the form of a thick bead, such as is used for the front sight of a rifle. It was found that a thin blade could not be seen sufficiently well. Also, the tip should be long enough to reach well into the lumen of the bronchus, which permits it to be plunged deeply into the growth. The perfection of this instrument has made possible much better results, more quickly obtained. For instance, the destruction of the tumor in Case 1, A. B., called for applications during two years. Case 4, which was the next one successfully treated, had only three treatments. On the other hand, she had a continuing bronchiectasis which might have been caused by stricture due to the too extensive destruction of the bronchial wall. Case 7, as has been noted, had a stricture following treatment. Case 8 had only four applications of the diathermy current. Case 9 needed six treatments. This tumor, however, was growing from the wall of the left upper lobe bronchus, three-quarters of an inch from the main bronchus, and was so difficult to visualize that the application of the current had to be proceeded with very cautiously. Case 10 had three applications of diathermy in the course of three weeks. At the end of that time the tumor completely disappeared. Two months later a bronchoscopy showed only a little dimpling of the mucous membrane at the site of the former growth. This is my most successful case up to the present time. It was possible with the instrument I now use

to get a perfectly accurate application of the electrode and to observe the destruction of the growth as it proceeded.

As I have already mentioned, the fact that these tumors grow outside as well as inside the bronchus, the question may well be asked How can one be sure that the outside part is destroyed? It will be recalled that when diathermy is applied, the current concentrates about the small electrode, heats the tissue in its neighborhood and tends to destroy the whole growth, not merely that portion within the bronchus.

REPORT OF CASES.

CASE 1.—A. B., white, female, aged 35, was admitted to Lenox Hill Hospital on December 12, 1926. Since December, 1922, she had been having attacks of pain in the chest, dyspnea and fever which had been variously diagnosed as pleural pneumonia and massive collapse of the left lung. X-ray finally confirmed this diagnosis. She had been bronchoscoped with negative findings.

At the time of her admission to Lenox Hill Hospital she was complaining of marked dyspnea and pain in the left side of her chest. She appeared acutely ill with temperature of 103.4°, pulse rate of 114, and respiration 28. She was cyanotic and very uncomfortable from the distressing dyspnea.

On examination, the physical signs were consolidation of the left lung. X-ray examinations were unsatisfactory, and reports were doubtful at this time. A provisional diagnosis of atelectasis was made. On December 18, a bronchoscopy was performed. The left main bronchus contained a tumor which almost filled the lumen. It was rough, vascular and friable. The appearance suggested malignancy.

The following microscopic report on the section was made: "The tissue fragments showed a malignant neoplasm composed of small hyperchromatic cells, containing scanty cytoplasm and relatively large nuclei which were either solid or vesicular. The outlines of the cells were poorly delineated, though the predominating cells appeared round. The cells were disposed in small solid alveoli, in diffuse masses and in multiple rows about central cores of vascular fibrous tissue. The peripheral cells of the alveoli were often elongated with their long axes directed at right angles to the stroma. The latter was generally sparse. In places it was vascular, while in other places it was dense and hyaline." A diagnosis of a malignant tumor, either carcinoma or endothelioma, was made.

The following is a summary of treatment used in this case: Beginning on December 29, 1926, and ending March 20, 1928, this

patient was bronchoscoped twelve times, each time the growth being partly coagulated with diathermy. On March 23, 1927, three radon seeds were inserted into the base of the growth. One of these was removed March 23rd. The other two could not be located. A total of nine millicurie was used. At the last bronchoscopy, March 20th, no sign of growth could be seen, the site being occupied by a small area of white scar tissue. It has been found possible in the treatment of more recent cases to diminish greatly the number of bronchoscopies and to shorten the period of treatment. It must be remembered that in this first case the treatment was experimental.

This patient has remained well up to the present time, having been bronchoscoped in April, 1932, for observation, over four years after the first observation of the growth. A recent report from her stated that she was in perfect health now.

CASE 2.—H. G., white, female, aged 17, was admitted to Lenox Hill Hospital on April 26, 1927, with a history of having had hemoptyses for six years. Except when troubled with bleeding, she had been in good health. X-ray films, made in January, 1924, showed a shadow in the right lower lobe, which suggested to the radiologists either an abscess or a tumor, perhaps benign in character.

An x-ray examination made in April, 1927, showed the same shadow in the right lower lobe. The radiologists felt that it had not grown in the interval of three years, and that it must be nonmalignant. A bronchoscopy, April 26, 1927, showed a large vascular tumor in the lower right main bronchus.

The biopsy report was: "Malignant tumor, probably carcinoma. Tumor emboli in blood vessels. The specimen in formalin consists of several small irregular fragments designated 'Tumor of right bronchus.' Sections of the tissue fragments show a malignant growth, the bulk of which is composed of small alveoli filled with rather small cells with poorly defined outlines and round or oval nuclei. The alveolar walls are usually thin and cellular. In one part of the tumor the cells take a deeper basic stain and appear somewhat larger than the rest of the cells. These cells are disposed in compact masses or in imperfect columns supported by strands of vascular stroma. Tumor emboli are present in several of the smaller blood vessels. The general structure of the tumor suggests a pulmonary origin."

This patient had four or five deep x-ray treatments in the summer of 1927 and thereafter no treatment. From then until December, 1931, she had no more hemoptyses. At the present time she continues to be in good health.

Because it was deemed impossible that the original tumor could have been malignant and the patient at the same time have remained in good health, the slides taken at the biopsy were re-examined by Dr. Grethman of Bellevue Hospital and Dr. James Ewing. They both confirmed the diagnosis of carcinoma from the slides. It is important that the diagnosis of carcinoma was made in this case. Such a diagnosis should not indicate pneumonectomy unless the other data concerning the tumor confirm such a diagnosis.

CASE 3.—A. J., aged 54, white, female, was first admitted to the Presbyterian Hospital on May 6, 1929; she was last admitted on August 15, 1934. Except for the symptoms of her pulmonary disease she had been always well. At the age of six she developed a cough. At the age of thirteen she had a hemoptyses. Then for the next forty years she had hemoptyses of varing severity necessitating treatment and hospitalization. She was bronchoscoped at the Presbyterian Hospital in 1929, and the right main bronchus was found blocked by a vascular tumor, of which a biopsy was taken. The diagnosis of carcinoma was made on this biopsy. A subsequent biopsy at Mt. Sinai Hospital, in 1932, showed the growth to be a nonmalignant adenoma.

At the time of her admission, in 1929, physical examination showed a chronically ill, thin woman coughing up small amounts of dark blood. The left lung was negative. There were the physical signs of complete consolidation of the right lung. A description of the section of the tumor was as follows: "Section shows connective tissue, a large portion of which is necrotic, appearing as homogeneous, acellular, pink strands, at the edges of which there is calcium deposit. The rest of the tissue is a fairly dense stroma in which there are a moderate number of blood vessels and which is everywhere infiltrated by collections of small cuboidal cells, which in places show a tendency toward alveolar arrangement. The nuclei of these cells are hyperchromatic. Mitotic figures are not seen. A few polymorphonuclear leucocytes and lymphocytes are scattered through the tissue. Diagnosis: Carcinoma of lung. Note: It is impossible to tell from this section whether this is a carcinoma or a mesothelioma of the pleura which has invaded a bronchus. The cells resemble mesothelial cells rather than epithelial cells, but in the absence of definite x-ray evidence as to primary pleural involvement this diagnosis does not seem justified." Then a year later, on October 29, 1930, it was agreed that the "specimen is a small round cell carcinoma of bronchial origin, a slow-growing neoplasm, in view of absence of mitotic figures."

In August, 1934, it was finally decided that the only course left to pursue was a pneumonectomy. For the first five days after the last admission, only a small amount of bleeding occurred, and the left lung was absolutely clear. The operation was performed on August 20, 1934. Since considerable difficulty was experienced in freeing the right lung, which was adherent to the parietal wall, diaphragm and adjacent pericardium, it was decided during the course of the operation merely to separate the adhesions, put a loose tourniquet around the hilum, and leave the completion of the pneumonectomy for a later date. During the first twenty-four hours postoperatively the patient's condition was remarkably good, blood pressure remained up, color excellent and breathing free and not labored. On the morning of the second day respiration became considerably more difficult with a pronounced expiratory grunt, moist räles at both bases. There was slight cyanosis. Her temperature gradually rose to 103°, the pulse rose to 130 and became thready and feeble. Throughout the day cyanosis increased, respirations became progressively more rapid and shallow. At 8:00 o'clock that evening Cheyne-Stokes type respiration set in, and at 8:45 she died.

On autopsy it was discovered that the entire lung was collapsed, measuring 12 x 10 x 8 cm. The external appearance of the organ was so distorted by collapse and fibrosis that only with great difficulty could the division of the lung into three lobes be demonstrated. There was a firm, raised, smooth nodule projecting into the right main bronchus covered by bronchial mucosa. This nodule was continuous with a tumor the size of a grapefruit which had compressed the middle and lower lobes as to completely obliterate them. The upper lobe bronchus was completely closed by pressure from the tumor and the lower lobe was occupied by many bronchiectatic cavities. The small portions of the middle and lower lobe not occupied with the tumor were occupied by bronchiectatic cavities. Microscopic sections made of this tumor showed it to be the same structure as sections taken at the previous biopsy.

Microscopic Examination. Tumor Nodule at Bifurcation of Right Bronchus.—The surface of the nodule is everywhere covered by a fairly thick band of fibrous tissue. In this at one side of the section there is a mass of dark purple calcified material. Here the collagenous tissue is widest, is relatively anuclear and has a homogeneous hyaline appearance. Farther along, the remnants of a columnar epithelial covering can be seen. Small vessels occur in this coat and some parts of it are more cellular than others. At one point the connective tissue is extremely thin and the underlying tissue is on the point of

breaking through. There are some small regions where the fibers are separated and the tissue seems edematous. At approximately the apex of the nodule there is a slight depression, from the bottom of which a broad band of dense connective tissue extends downward into the tumor, growing narrower as it goes. Beside it and reaching almost to the surface there is a small, thin-walled artery distended with blood. Beneath this fibrous coat lie masses of tumor cells in a sparse, loose connective tissue stroma. The arrangement of the neoplastic cells varies remarkably, sometimes even in the same field. In some places the tissue has an adenoid appearance with the cells arranged circularly around a definite lumen. Elsewhere it consists of large irregular masses or cords loosely surrounded by fine connective tissue membranes. In still other places it appears as delicate branching and anastomosing strands. The greater portion of the tumor seems to be growing free in alveolar spaces. The intervening connective tissue strands contain capillaries and seem to be separate structures rather than a tumor stroma. In addition there are many large areas of degeneration from which all nuclei have disappeared and only a uniformly pink or violet staining area in which, although necrotic, the original architecture can still be seen. Other regions are composed of amorphous, closely packed masses of rounded tumor cells accompanied by little or no fibrous elements. In these places there are usually foci of nuclear loss and beginning necrosis. Occasional small hemorrhages can be seen. The individual cells of the tumor vary only slightly, although in the larger masses and cords they tend to be larger and their cytoplasm to be less acidophilic. Likewise, their nuclei are larger and lighter. Elsewhere they have small round or oval, very dark nuclei. In those which are less dark and in which separate chromatin masses can be made out, the latter tend to have a somewhat radial distribution suggestive of the plasma cell. The cytoplasm is homogeneous, pale pink and is not abundant. The cells are rounded or irregularly polygonal, depending on the pressure of neighboring cells. No mitotic figures are seen and there is no keratinization. There are several moderately large thrombosed vessels in the neoplastic area.

Elastic Tissue Stain.—There is no elastic tissue in the fibrous septa resembling alveolar walls seen in the H. and E. stain between the smaller masses of tumor cells. Elastic fibers in the walls of the arteries are well stained.

Mallory Connective Tissue Stain.—There is an extremely sparse fibrous stroma. Relatively large areas are practically devoid of connective tissue. In some areas of more abundant fibrous tissue the

tumor seems to be invading the collagenous tissue rather than the latter acting as a support for it. At one point the tumor cells have broken through the fibrous coat separating them from the lumen of the bronchus.

Tumor: A.—The pleomorphism of the tumor is again evident here. There also are large regions of degeneration. In addition there is a small area of calcification in which true bone formation has occurred with beginning development of a marrow cavity. Near this is a broad band of fibrous tissue sparsely infiltrated with polymorphonuclears, lymphoid and mononuclear cells, and containing occasional deposits of greenish-black pigment. Small masses of tumor cells lie in spaces in the collagenous tissue.

Mucicarmine Stain.—There is no mucin production by the tumor cells. The alveolar structure is well brought out.

Elastic Tissue Stain.—The absence of elastic fibers in the connective tissue surrounding small masses of tumor cells is again evident, although elastic tissue in the arterial walls is well stained.

B. The tumor here is essentially similar to that in the previous slides. There is a large degenerated area in which lie several relatively large arteries containing thrombi.

Foot Bielschowsky Stain.—Around the masses of tumor cells there is a coarse reticulum from which finer fibers run into the mass and between individual cells.

C. This is a section from the base of the middle lobe. The pleural side consists of loose fibrous tissue through which run large and small vessels engorged with blood. Extensive hemorrhage has occurred into the greater portion of this fibrous tissue, but there is little cellular infiltration. The outermost portion contains some fibrin and numerous bacteria, but even here there is no cellular reaction. Some of the inner portions of this fibrous tissue are hyalinized, while others appear as a homogeneous pale pink anuclear material. There are occasional areas of elastic tissue scarring in the fibrous portions. There are a few small deposits of greenish-black pigment granules. The rest of the section, inside the fibrous region, consists of degenerated tumor tissue, all of which, including the nuclei, takes a bluish pink stain. Along the outer margin of the tumor there are deposits of a dense dark purple material lying between or replacing the tumor cells.

Base of Right Lower Lobe.—The inner portion of the section consists of tumor, largely degenerated, in which are several large

thrombosed arteries, frequent foci of calcification, and two moderately large spicules of true bone. The tumor cells are closely packed together or strung along branching strands of connective tissue. As in previous sections, many seem to be growing in alveoli. In the fading nuclei the previously mentioned radial distribution of chromatin masses, which gives them their resemblance to lymphoid cells, is clearly seen. Surrounding the tumor mass there is a smooth, regular capsule of dense connective tissue which, over an appreciable portion of its outer surface, is covered with a single layer of low cuboidal cells. A few small collections of tumor cells occur outside of this capsule, but there is no change in the character of these cells. Opposite the capsule and separated from it by an open space is a similar layer of uniform connective tissue. Immediately outside of this there are a few more masses of tumor cells, and then a broad region of loose, irregular, collagenous tissue, much of which appears hyaline and through which run many engorged vessels of varying size. There are occasional collections of lymphocytes, much hemorrhage, some calcification and frequent deposits of anthracotic pigment. Caught in the strands of the outermost portion there are numerous masses of bacteria.

Gram Stain.—The organisms seen in the H. and E. stain are chiefly gram positive cocci, but large gram positive bacilli are also numerous.

Tumor Capsule.—On the inner side there are a few bits of tumor. Near by is a relatively large area of bone formation. The rest of the section consists of fairly dense, hyalinized, interwoven strands of collagenous tissue. Included are two large arteries, the walls of which are thickened, almost to the point of occlusion.

Right Upper Lobe.—Along the pleural surface there is a small coating of finely granular material containing occasional fibrin strands. Beneath this the pleura is much thickened, edematous and contains many small engorged vessels. The parenchyma is greatly distorted. The alveoli are compressed and their walls thickened by connective tissue. Most of the alveolar spaces are filled with extravasated red blood cells. Many of them also contain numerous large mononuclears, often filled with greenish-black pigment. In the neighborhood of the dilated bronchi there are large areas of fibrosis, many composed of loose and edematous tissue. In these areas only an occasional remnant of an alveolus can be seen. Everywhere throughout the section vessels are distended with blood. The bronchioles are immensely dilated and are for the most part empty, but some contain a deep blue staining coagulum. They are lined by tall columnar

epithelium. Metaplasia of the epithelium has occurred in several. The connective tissue around many of the bronchioles is densely infiltrated with lymphoid cells and polymorphonuclears. Partial calcification has occurred in the bronchial cartilage.

Final Note.—An interesting case of a woman, aged 54, who had had large hemoptyses for five years. Four years ago a diagnosis of carcinoma of the lung was made from a piece of tissue having been removed at the time of bronchoscopic examination. In the lower and middle lobes of the right side is a tumor infiltrating the lung extensively, and occluding the bronchi of these two lobes and extending into the bronchus of the upper lobe. The tumor is composed of small cells of uniform size, having infrequent mitoses and not tending to form glands nor to produce mucin. The morphology of the cells suggests that the tumor grew slowly. The origin of the tumor is probably not from the larger bronchi but from the more distal portion, as for example, the ductus alveolarus. No metastases were found in the opposite lung or in the bronchial lymph glands. This case was presented at conference November 22, 1934, as an example of the group of cases which Dr. Stout calls "tumors" of the bronchus. It would seem to be a malignant tumor which arose from what is called "Intrabronchial Polypoid Adenoma," by Reisner, Archives of Surgery, Volume 16, 1201, and "Adenoma," by Wessler and Rabin, American Journal Medical Sciences, volume 183, 1932, 164. These tumors have also been called "basal cell carcinomas," arising from mucous gland ducts, at Johns Hopkins Hospital, and also "carcinoids."

These tumors, of which some thirty have been reported, form a clinically recognized group, which begin as smooth polypoid adenomata, partly occluding a main bronchus, and in most cases remain benign and noninfiltrating over a period of months or years. They do not, even after becoming invasive, involve the regional lymph glands nor do they metastasize.

The origin of the tumor is not entirely clear. There are three possibilities: bronchial epithelium, mucous glands, and duct epithelium of these glands. The fact that, in the early stages at least, these tumors are covered by an intact bronchial mucous membrane argues against this as the origin. Furthermore, they do not characteristically form mucus as would be expected if they originated in the mucous glands of the bronchi. This leaves the epithelium of the ducts of these glands as the most probable origin. The occurrence of cilia with basal corpuscles in one of the sections in this case would seem to exclude an alveolar origin.

CASE 4.—A. D., a thirty-three year old Russian Jewish housewife was first admitted to the Presbyterian Hospital on October 16, 1929. The patient started having trouble about March, 1928, when she developed a productive and persistent cough. The sputum, varying from none to 250 cc. in twenty-four hours, was of bad odor. Several small hemoptyses occurred.

Physical examination made on this moderately obese woman revealed that her trachea was in midline with no tug, and that the thorax was symmetrical. The right lung was clear, but the left one showed diminished expansion with numerous räles and ronchi. The signs suggested limited cavitation in the left lower lobe.

On November 6, 1929, a bronchoscopic examination revealed a vascular tumor just beneath the left upper lobe bronchus. A pathologic report on this tissue pronounced it a carcinoma of unknown origin, poorly differentiated, with no mitoses. The growth was destroyed by diathermy in the course of three treatments. Thereafter her symptoms were those of an extensive bronchiectasis. The diagnosis at the later admissions was "bronchiectasis of the left lower lobe due to carcinoma of the bronchus." On June 13, 1933, the following x-ray report was made: "Film of the chest following the instillation of lipiodol into the left lower lobe bronchus shows the lipiodol outlining a few saccular cavities here suggesting a bronchiectasis. The lumen of the main bronchus is not completely filled, nor is the entire left lower lobe bronchial tree. At present there is no evidence of an atelectasis of the left lower lobe to suggest that there has been a bronchial occlusion with possible secondary bronchiectasis. The two year course of this case makes one wonder if it is a true bronchial carcinoma."

The treatment of this case had consisted of diathermy, implantation of radon seeds, postural drainage and bronchoscopic drainage. Although clinically the patient had gotten no worse, as would have been expected in a poorly differentiated neoplasm of presumably five and a half years' duration, on account of the bronchiectatic symptoms which had persisted in spite of numerous bronchoscopic treatments, it was finally decided to do a lobectomy. This was entirely successful in restoring her to robust health. Examination of the tissue found in the lobe removed at the operation showed no evidence of the original growth.

In May, 1935, this patient continued to be in good health.

CASE 5.—A. Q., white, female, aged 40, was admitted to the Lenox Hill Hospital on August 21, 1930, with a complaint of loss

of weight (twenty pounds in six months), and loss of appetite for ten months. She had been treated for various stomach ailments for ten months but to no avail.

Fluoroscopic examination revealed a small shadow in the right lung. Physical examination of this small, poorly nourished woman proved to be negative except that the tactile fremitus was decreased slightly over the anterior chest on the right side. No räles were audible and breath sounds were normal.

On August 21, 1930, a bronchoscopy revealed the following: Below the level of the right upper lobe bronchus there was a tumor mass, shiny, irregular and polypoid in appearance, which apparently obstructed the entire lumen of the bronchus. A biopsy specimen, causing profuse bleeding, was reported as follows: Section of one of the fragments showed a carcinoma composed of acini, usually of small size, intermingled with small solid islands of cells. The cells lining the acini were usually low columnar in form and one to several rows thick. The solid structures were formed by small polyhedral cells with deeply staining nuclei and sparse cystoplasm. The stroma of the tumor was relatively scanty, although it varied in amount in different areas. It was, for the most part, poor in cells, though well vascularized and often showing considerable edema. In small localized areas in was myxomatous. Section of the other fragment showed a carcinoma apparently composed exclusively of small glandular acini, the cells of which were larger and more chromatic than those of the first described fragment. These structures were crowded close together, being separated by strands of vascular fibrous tissue. Diagnosis: Adenocarcinoma of the bronchus.

From August, 1930, to November, 1930, the patient was bronchoscoped, with coagulation of the tumor diminishing perceptibly the size of the mass.

A year later, in November, 1931, when the patient was readmitted to the hospital, her general condition had improved. She had been raising a little mucus upon arising, but there had been no excessive coughing during the day and no pain in her chest. X-rays of the chest at this time showed a large triangular area filling the region between the right heart border and the right side of the diaphragm; three radon seeds were still visible from the apex of the triangle; the upper three-fourths of the right lung field and the entire left appeared normally clear.

The patient was bronchoscoped at frequent intervals during the next four months. The bronchus was rough looking but gave no

evidence of malignancy. A report on a biopsy taken diagnosed the tissue from the bronchus as a chronic inflammation.

In October, 1934, the patient was bronchoscoped again, at which time the inflammatory tissue was again noted near the cardiac branch, with a scanty amount of discharge, but it did not have the appearance of a recurrence.

The patient, who was at the hospital recently, was feeling fine and had no complaints.

CASE 6.—L. K. was admitted to Lenox Hill Hospital October 12, 1930, complaining of dyspnea and cough of two weeks' duration. She had had pain over her left chest for three days, which was not aggravated by breathing. Her cough was unproductive.

Examination showed that the left side of the chest expanded very little, with a dullness of the whole upper half and flatness at base, both anteriorly and posteriorly. Absent fremitus and absent breath sounds at the base of the lung were noted. In the upper half of the chest anteriorly was a high pitched distant tympanitic percussion sound. Breath sounds had an amphoric quality. The same existed posteriorly but breath sounds were distant. No räles were present anywhere. The right lung was over-aerated and hyperresonant with exaggerated vesicular breath sounds. A tentative diagnosis was made of massive collapse of the left lung, due to the occlusion of the left bronchus by a possible carcinoma. X-ray examination showed atelectasis of the left lung. Wassermann test was negative.

A bronchoscopy performed shortly thereafter revealed that the left bronchus was completely closed by a mass of tumor tissue which sprang from its wall and also involved the trachea for an inch above the bifurcation. The right bronchus was also closed by tumor masses. The tumor mass, which was vascular, friable and subject to bleeding when touched by the tube, appeared to be malignant.

Two days after the bronchoscopy a second x-ray examination showed that the entire left lung was much clearer and also normally aerated, contrasted with the atelectasis of the previous x-ray examination four days before.

Examination of the biopsy taken at the first bronchoscopy showed that the specimen had a stroma of very dense hyaline connective tissue, in which there was a most intense round cell infiltration. One small portion of the section had a new growth composed of polyhedral cells arranged in small acini. The individual cells were intensely hyperchromatic; the nuclei were central. A classification of

the tumor type could not be made on the material submitted at that time, but the diagnosis given as malignant tumor, most probably small celled carcinoma.

The patient left the hospital without treatment. She was not seen again until June 30, 1931, when a follow-up x-ray revealed no roentgen evidence of the lesion in the left bronchus.

On March 21, 1934, the patient, readmitted to the hospital for a bronchoscopic examination, was feeling fine with no complaints of cough, loss of weight, fatigue or chest symptoms. The bronchoscopy showed nothing pathologic in the bifurcation or right main bronchus. Near the mouth of the left main bronchus there was a fibrous band stretching across the lumen posteriorly, forming a sort of shelf, with a little canal running under it. The fibrous adhesion was ruptured, and a small spot of what appeared to be granulation tissue was seen on the posterior wall. The tumor in the bronchus of this patient had disappeared without treatment.

CASE 7.—B. B., white, male, aged 13, was admitted to the Lenox Hill Hospital on February 5, 1931. Eleven days before admission he had taken cold, accompanied with pain in the legs and a temperature of 101. Two days later he had a temperature of 104, a cough with considerable expectoration, at times blood streaked, but no pain in his chest. His case was diagnosed as a left lobar pneumonia, a day following the crisis of which he had quite a hemorrhage. X-ray pictures at this time showed a uniform density of the lower half of the left chest; the heart was drawn to the left; the upper left chest was also cloudy, though not to the same degree. The diagnosis, based on the x-ray examination, was the presence of pleural fluid in the lower left chest. Three days later the patient was admitted to the hospital. The same vomiting of blood which had occurred a few days before admission continued several times after admission.

The patient was observed for a possible empyema or foreign body. He began to improve and continued to do so. Though bronchoscopy was considered at this admission, it was not done. The lesion in the left chest showed considerable clearing up in the upper half, and the lower half remained uniformly dense.

Over a year later, on June 27, 1932, he was readmitted to the hospital with a recurrence of his symptoms of cough, temperature and pain in the left chest of eight weeks' duration. X-ray at this time revealed a recurrence of the processes which had been present before. A bronchoscopy performed then revealed a tumor protruding from the left and posterior wall off the bronchus, partially block-

ing the lumen; the tumor was rough, firm and vascular; the mucous membrane below and above it was normal. The microscopic examination of the biopsy taken showed: "A malignant epithelial growth, the bulk of which is situated in the deeper part of the tissue. In this region the structure consists of compact nests of rather small rounded or polyhedral cells supported by a relatively small amount of dense fibrous tissue which is poor in cells and blood vessels. The cell nuclei are round and usually vesicular, and the cytoplasm is scanty and moderately acidophile. Here and there are noted larger cells containing a single nucleus. More superficially, the cells become smaller, more compressed and grow in narrow and often irregular and ramifying strands which show a tendency to retract from the supporting stroma. Some of these cell strands extend into the mucous membrane, but have no connection with the surface epithelium. The latter, however, in places is completely eroded away. In parts of the tumor are noted round or slit-like spaces bordered by one or several rows of tumor cells. Definite glandular structures, however, are not observed. Neither intercellular bridges nor epithelial pearls are demonstrable. The stroma shows rather slight round cell infiltration and a few small scattered hemorrhages. Careful search fails to reveal the presence of mitotic figures.

During the next six months the patient was bronchoscoped about eleven times. Diathermy very quickly reduced the growth to a roughening on the posterior wall of the bronchus about the mouth of the upper lobe bronchus and extending a short distance into the lower lobe. Before long the growth disappeared entirely. Bronchoscopy in October, 1932, revealed a constriction of the lumen of the left bronchus, below the level of the upper lobe bronchus, and a little mucopurulent secretion in the neighborhood of bifurcation. X-ray pictures at this time indicated considerable bronchiectasis in the mid-left chest.

The patient was bronchoscoped three times in 1933, in February, April and July, at which times no pus or neoplasm was seen. The condition of the lung seemed improved, though there was still some congestion in the main lower lobe bronchus.

The last of December, 1933, the patient was readmitted to the hospital with chills, cough, dyspnea and pain in the chest. A diagnosis of occlusion of the left main bronchus with atelectasis of the entire left lung was made. Bronchoscopic examination proved that there was no new growth but that the lower lobe bronchus was a dense mass of scar tissue through which were two openings, the anterior one discharging pus and the posterior one coming from the

first dorsal branch which was fairly well open. In the constricted lumen of the brain bronchus, represented by the anterior opening, could be seen the granulation tissue. An attempt was made with the dilator to dilate the bronchus, which was unsuccessful. X-ray examination shortly thereafter indicated a diagnosis of bronchiectasis with considerable pneumonitis present at the left base surrounding what appeared to be multiple small cavities.

Subsequently the patient developed small abscesses below the level of the stricture in the left lower lobe and was operated upon. A rib resection with drainage of small multilocular abscesses was done and a biopsy of the lung was taken. The pathologic report was chronic suppurative inflammation of the lung with bronchiectasis and chronic interstitial pneumonitis; there was no evidence of neoplastic disease.

On October 15, 1934, the patient was again operated on for a chronic fistula, followed by a lobectomy. He died the following day. The pathologic report at the time of lobectomy was chronic pneumonitis with bronchiectasis. There was no evidence of malignant disease.

CASE 8.—R. F., white, female, aged thirteen, was admitted to the Lenox Hill Hospital on January 4, 1933, with a complaint of weakness, frequent colds, a persistent cough with no expectoration, hemoptyses, intermittent attacks of fever but no loss of weight. (Her family history revealed carcinoma in the case of her grandmother, her father and one aunt.) About a year before admission to the Lenox Hill Hospital the patient had been admitted to St. John's Hospital, Long Island City, where her condition had finally been diagnosed as tuberculosis. X-rays were taken and a left pneumothorax had been performed. She had been discharged from St. John's in February, 1932, with a very poor prognosis. In March she went to the Department of Health for re-examination, where she was told she did not have tuberculosis and was sent to Bellevue Hospital for diagnosis. There she was bronchoscoped four times. Two biopsies were reported as carcinoma of the bronchus and two as polyp of the bronchus.

On admission to Lenox Hill Hospital, examination showed a few räles in the upper part of the right chest posteriorly with no evidence of previous pneumothorax; the remainder of the chest was clear. A bronchoscopy was performed at this time, and in the left main bronchus, above the level of the upper lobe bronchus on the anterolateral aspect, there was a small nipple-like tumor. X-rays

taken then revealed that there was "an indefinite density in the left lower half of the chest with pulling over of the cardiac shadow and elevation of the left side of the diaphragm. Lung tumor with bronchial obstruction would be suspected as a cause of the appearance presented." Microscopic examination of the biopsy taken at the bronchoscopy showed a fibrous nodule, probably inflammatory. Another biopsy, taken a week later, showed a section which suggested a small-celled carcinoma of the bronchus.

In the next three months diathermy was applied to the tumor through the bronchoscope three times, with the result that the tumor mass disappeared and was replaced by a scar, leaving the bronchus wide open.

Bronchoscopies were performed at irregular intervals, and, when examined on August 9, 1934, there was no sign of a recurrence. However, a lipiodol injection by the drip method showed that the patient had an extensive bronchiectasis, which was concluded to be dry, since the patient had no symptoms of cough or expectoration.

CASE 9.—M. S., white, male, aged 33, was first admitted to the Presbyterian Hospital on September 18, 1934, with a chief complaint of cough and fever of thirteen months' duration. His illness originated with a mild cold, which after exposure had become suddenly worse with chest pain, cough with a little sputum of forgotten type and fairly severe fever. At this time the local doctor diagnosed the case pneumonia and sent him into St. Luke's Hospital, Utica, N. Y., for three weeks. The pneumonia was a lobar one of the left upper lobe with perhaps some involvement of the right lower lobe. Type IV pneumococcus was found. On discharge the patient still had considerable cough, raising two ounces of sputum, and with spells of fever every few days. Twelve months ago the patient, weighing eighteen pounds less than usual, went home with the spells of fever and cough continuing; he was also suffering from severe night sweats. Sputum was scanty with only an occasional small greenish bit of mucus, not foul, no blood ever. Eleven months ago x-ray pictures were taken at St. Luke's, in Utica, which were interpreted as tuberculosis. No sputum examination was made. Ten months ago, as his symptoms continued, the patient went to Broadacres Sanatorium in Utica, where he remained six months. Two more sputum examinations before admission were negative, and in the hospital at least a dozen more were negative, and two guinea inoculations were unproductive. X-rays of his chest were said to show a "honey-comb shadow" in the left upper lobe. Sanatorium doctors apparently disagreed as to diagnosis and never would call it tuberculosis. During

the stay the patient's weight returned to normal. The cough improved, but the x-ray shadow changed little. Fever became less frequent, coming about every two weeks and lasting only a few days, still of spiky type. The patient had no more night sweats. For the last four months the patient had been in the Catskills, pretty much in bed. The same fever spells continued and became more frequent, coming every few days or lasting fairly steadily. He had no more night sweats and no loss of weight. Sputum was negative again two months ago. The sputum increased slightly in the last month before admission. As the bout of fever comes on, the patient had more cough and more sputum, but the sputum comes more at night than in the morning. The patient finally came to the P. H. because of his failure to improve. The patient reported that doctors had always commented that no one had ever recorded hearing räles in his chest.

Examination of his lungs showed: "Anteriorly over an area about 6 cm. in diameter, extending from the clavicle to the fourth rib and from the sternum out 6 cm. laterally, there is dullness, diminished but normal B. S. and decreased T. F. and V. F. Above the clavicle and lateral to this area all is normal. Posteriorly over a corresponding area (interscapular and suprascapular, but not to true apex) the V. F. and T. F. are decreased but B. S. and resonance are normal. No räles heard. Rest of lungs clear. Both bases level and descend well. The impression gained was a possibility of lung abscess or interlobar empyema." At the same time stereoscopic chest films showed an extra density in the left middle lung field which stereos anteriorly. There was a suggestion of a cavity in the center, but no definite fluid level was seen; the findings were thought to be pretty definitely those of a lung abscess. Two weeks later it was decided that the x-ray studies confirmed the history diagnosis of lung abscess, placing it in the left upper lobe; the process was supposed to be in such an advanced chronic stage that spontaneous healing seemed impossible; drainage seemed necessary, perhaps to be followed eventually by a partial pnumonectomy or lobectomy. It was felt, however, that the possibility of a lung abscess in association with bronchial neoplasm could not be definitely ruled out without bronchoscopy, a bronchoscopy was performed. The bronchoscopy revealed that the right side was negative; a cluster of granulation tissue or soft neoplastic mass was seen coming out of the mouth of the left upper lobe bronchus, with no pus descending from this bronchus; no pathology or pus was seen in the lower lobe bronchi. The biopsy report on the specimen taken at this bronchoscopy was as follows: "The specimen consists of a very small mass of soft pink tissue partly covered by

mucous membrane. It measures 7 x 5 x 4 mm. The mucous-covered surface is slightly nodular. The cut surface is soft. Specimen fixed in Bouin-embedded in toto. Sections of the pieces of tissue removed show that it is almost completely filled by a tumor. This is composed of slender, solid, anastomosing strands of cells which are rather small, cuboidal, have centrally placed nuclei, and faintly acidophilic, finely granular cytoplasm. The cords of tumor cells are separated one from the other by rather delicate strands of collagen fibers in which the blood vessels are found. No mucin is seen in the cells, and there seems to be no tendency to form acini. A small amount of the bronchial musculature is present, and along one margin a little bronchial mucous membrane. The growth seems to lie deep to the small fragment of muscle which is present in the section. No mitoses are seen. This is the type of tumor of the bronchus which tends to grow slowly, project into the lumen, causing partial or complete obstruction with resulting bronchiectasis and infection. It tends slowly to penetrate the bronchial wall and to grow to a limited extent in the tissues outside of it, but so far as our experience extends, it does not metastasize. No satisfactory name has been generally accepted. Both the terms 'adenoma' and 'carcinoma' have been used, and at the present time we are using the noncommittal term 'tumor.' If the case is operable, a cure may be effected by lobectomy or pneumonectomy. Diagnosis: 'Tumor' of the bronchus."

The patient came in for ten subsequent bronchoscopies during the next six months to date, at the first one of which it was discovered that the soft and more or less pedunculated tumor dammed up a considerable amount of pus, which was released when several bits of the tumor were bitten away. At the next seven bronchoscopies the tumor was fulgurated with diathermy, thereby completely destroying the growth. He was bronchoscoped several times afterwards for observation. On his last admission, eight months after his first admission to Presbyterian Hospital, he appeared to be in very excellent condition, with no cough, no sputum and no hemoptyses. The breath sounds over the left chest were coming through quite distinctly, indicating that the lung had re-expanded.

Case 10.—G. E., white, female, aged 26. This patient had had cough and shortness of breath for a year. Occasionally there were acute febrile attacks, due seemingly to complete plugging of the right bronchus and collapse of the lung. These attacks had been increasing in frequency and in severity.

On June 14, 1934, this patient was bronchoscoped. A soft tumor was found which appeared to have a tendency to be pedunculated.

A biopsy was taken at this bronchoscopy. The pathologists who examined the sections differed greatly in their reports, some insisting that it was a carcinoma, on the basis of which report the thoracic surgeons urged a pneumonectomy. Consent was given to try the effect of diathermy.

On June 28, 1934, July 5, 1934, and in July, 1934, she was bronchoscoped and diathermy was used.

On September 25, 1934, she was bronchoscoped again, at which time no remains of the tumor could be seen.

103 East 78th St.

Abstracts of Current Articles.

NOSE.

Pyocele of the Frontal Sinus (Sur les empyèmes enkystés du sinus frontal).

Prof. A. Laskiewicz (Poznan), Rev. de Lar., 56:561 (May), 1935.

Reporting two cases of thick-walled cysts of the frontal sinus lined with ciliated epithelium and filled with pus. These cysts were firmly attached to, and had thinned both anterior and posterior walls of the sinuses with strong fibrous adhesions; marked external edema was present, with much pain and tenderness. Radiographic findings resembled those of mucocele.

Complete extirpation by external radical operation was curative, without recurrence, in a few weeks. Such cysts are apparently due to closure of the nasofrontal duct in the presence of a bloody exudate with low grade infection. Symptoms include bulging downward of the floor of the sinus over the inner canthus, flexibility of the thinned anterior wall of the sinus under digital pressure, with inability to probe the nasofrontal duct and absence of pus in the nose. Severe frontoparietal pain becomes constant.

FENTON.

Histopathology of Septal Cartilage After Submucous Resection (Nota istopatologica sul comportamento della cartilagine del setto nasale nelle resezinni sottomucose).

Gianni, O. (Parma), Arch. Ital., O., R., L., 47:487 (July), 1935

Resecting the septal cartilage in dogs, Gianni found that no proliferation of cartilage ever occurred submucosally, but that firm fibrous tissue grew between the flaps from the perichondrium. Removal of one mucosal flap prevented the formation of this fibrous layer and tended to produce perforation.

FENTON.

Malignant Tumors of the Nasal Mucosa.

Price, L. Woodhouse, J. of Laryng. and Otol., L:153-184 (March), 1935.

The author recounts a series of malignant tumors of the nose and the accessory sinuses, and attributes to their particular anatomic relationships their high mortality rate. He discusses the clinical diagnosis and recommends the cytologic examination of material removed by aspiration. He describes the pathology of these growths

and proposes a classification based upon the nature of the epithelium from which they arise. Invasion of extranasal tumors into the nose is also discussed, and the complexity of paradental tumors is shown to be attributable to their origin from multipotential cells derived from the anlage of the enamel organ.

PHARYNX.

Syphilitic Angina With Tonsillo-palatine Plasmocytoma (Angini sifilitica con plasmama tonsillo-palatino).

Scalzitti, M. (Rome), Valsalva, 11-375 (July), 1935.

Reporting a case of bilateral pinkish-gray swelling and stiffening of the pharyngeal pillars, uvula and tonsils, with difficulty in swallowing and persistent otalgia, in a man of 38, Scalzitti found uniform connective tissue hyperplasia with marked plasma cell infiltration throughout. With the Levaditi stain, the spirochaeta pallida could be demonstrated throughout the parenchyma, clinching the diagnosis of secondary lues.

FENTON.

Malignant Tumors of the Nasopharynx.

Heine, Lyman H. (Boston), Arch. Otolaryng., 22:51 (July), 1935.

Nineteen cases of malignant tumor of the nasopharynx encountered at the Massachusetts Eye and Ear Infirmary from September 1, 1933, to September 1, 1934, are reviewed.

From this number, a group representative of the various types of neoplasm rather commonly located in this region was selected. Data on the cases have been presented in the order of the frequence of occurrence of the lesions in the series, along with a short discussion of the pathologic changes, treatment and follow-up in a few of the cases.

Reticular cell sarcomas have been described and separated from the old generalized grouping of lymphoblastoma.

The following conclusions suggest themselves:

1. The nasopharynx bears a rather fertile soil for the development of the various types of neoplasm.

2. Any abnormality in the appearance of the tissue in this region is worthy of biopsy.

3. Tumors arising from the different kinds of epithelial covering over the vault of the nasopharynx are different, both pathologically and clinically, from most other epithelial tumors and evidently deserve some special classification.

4. Reticular cell sarcoma occurs in the nasopharynx and should not be confused with lymphoblastoma.

5. The results in this series are in support of radiation therapy, but the follow up was limited to a period too short for conclusions as to the permanency of results.

TOBEY.

LARYNX.

Clinical Aspects of Laryngeal Cancer (L'aspect clinique du cancer du larynx).

Prof. G. Portmann and J. Herson (Bordeaux), Rev. de Lar., 56:525 (May), 1935.

Analyzing 169 cases, the authors state that 63.6 per cent are characterized clinically by hoarseness or by glandular invasion, and these usually wait too long before seeking help. Since pain characterizes the rarer forms—dysphagic or otalgic—these latter consult the laryngologist reasonably early. Therapeutic insufficiency in late cases, and the mediaeval destructiveness of our methods, surgical or by radiation, reflect our ignorance of the causes of malignancy. We have, however, no choice and must use these methods. Training people generally, and family physicians in particular, never to neglect chronic hoarseness after forty, and always to investigate glandular enlargement, are consequent upon study of the statistics which form the bulk of this valuable contribution.

The cases were located as follows: Vocal cord, 13; ventricular band, 12; both, 8; anterior commissure, 3; vocal cord extending to aryepiglottic fold, 2; ventricular band extending elsewhere, 6; epiglottic, 23; epiglottic extending elsewhere, 22; arytenoid, 7; arytenoid extending elsewhere, 13; aryepiglottic fold, usually extending, 6; pyriform sinus, 9; same, extending, 5; half the larynx involved, 32; advanced, diffuse, 14; total, larynx proper, 44 (25.2 per cent); pharyngolaryngeal, 125 (74.8 per cent). Loss of weight, due to autointoxication and dysphagia, is the most frequent general symptom.

FENTON.

Unknown Factors in Pericarotid Sympathectomy for Tubercular Laryngitis (Incognite delle simpaticectomie pericarotidee nella cura delle laringiti tubercolari, ecc.)

Prof. E. Rubaltell (Padua), Arch. Ital., O., R., L., 47:499 (July), 1935.

Recognizing the manifest improvement due to improved circulation brought about by pericarotid sympathectomy in the dysphagia of laryngeal tuberculosis, the author demonstrates that it

is dangerous to practice this resection at the carotid bifurcation because of damage to the carotid glomus, which is thought to assist in controlling and regulating breathing. He would limit perivascular sympathectomy to the superior and middle laryngeal and superior-thyroid arteries, or perhaps a short distance at the beginning of the external carotid. He feels that unless it can be proven that reflex central stimulation is added to the known peripheral vasodilatation by this procedure, such a difficult and dangerous method would better be abandoned in favor of recognized chemical, thermic or mechanical procedures.

FENTON.

Contact Ulcer of the Larynx.

Jackson, Chevalier, and Jackson, Chevalier L. (Philadelphia), Arch. Otolaryng., 22:1 (July), 1935.

Contact ulcer of the larynx is a disease characterized by a distinctive location, etiology, pathologic process, course and interminable duration unless it is recognized and properly treated.

It is usually overlooked; when seen, it is usually mistaken for cancer or tuberculosis.

The exciting cause is vocal abuse. The perpetuating cause is necrosis of the tip of the vocal process of the arytenoid cartilage located in the bed of the ulcer.

The diagnosis is made by mirror examination, by direct laryngoscopy and, rarely, by biopsy.

The regimen of silence is the most important part of treatment. If the talkative patient cannot be controlled he will frustrate all efforts to cure the condition. Excision of accompanying granuloma is sometimes necessary, and in some instances the ulcer itself may require excision. These measures, however, must be carried out with extreme neatness and delicacy, or irreparable damage may be done. Silver nitrate and other similar local applications are worse than useless.

TOBEY.

The Behavior of the Subglottic Pressure During Phonation (Sul comportamento della pressione sottoglottica durante la fonazione).

Cornelli, G. (Milano), Arch. Ital. di Otol., Rino. and Laring, 46:701-719 (October), 1934.

The author investigates the air pressure in the subglottic space during phonation, experimenting on a patient who had a (post-laryngofissure) tracheal fistula.

Graphs were taken of the subglottic pressure during the phonation of various types of words. These graphs reveal three characteristic curve changes which take place: (1) at the beginning of phonation; (2) during the period of maximum pressure, and (3) during the preparatory or latent period.

The first change in the curve is always constant, and it indicates the minimum limit of pressure for each vowel or consonant, which will always be reached whether the vowel or consonant is either at the beginning or in the middle of the word. This coefficient is influenced by the density of the voice.

The second change represents the period of maximum pressure, and it always varies according to the intensity of the sound and the location where the accent falls on the word. Therefore, this curve change is constantly proportioned to the sound intensity and moves in the curve according to the position of the accent.

The third, latent or preparatory, period varies at all times. It represents the time which elapses between the beginning of the increase in pressure of the breathed air and the emission of the voice. In reality this period is the pressure limit which is in relation to the rapidity with which the thoracic cavity contracts.

SCIARRETTA.

EAR.

Treatment of Thrombosis of the Lateral Sinus.

Meltzner, Philip E. (Boston), 22:131 (Aug.), 1935.

Symptomatically it is impossible to distinguish between sinus thrombosis and phlebitis.

In a total of 4,961 cases of resection of the mastoid, the diagnosis of sinus thrombosis was made in 161 cases, indicating that it occurred once in approximately 31 cases. Sinus thrombosis is the most frequent complication of mastoiditis, the greater number of cases occurring in association with acute otitis. It occurred more often in patients between eleven and twenty years of age, the curve of incidence tapering off sharply at both extremes of age.

Metastasis was an uncommon complication before ligation of the jugular vein. Involvement of the joints was the most common complication, the greatest number of cases occurring after ligation and treatment of the sinus. Involvement of the joints adds to the seriousness of the disease as it may prove to be the focus which continues to feed organisms into the blood stream.

Blood cultures are of value as an aid in establishing a diagnosis or at least in focusing attention on the lateral sinus. In the presence of continuing symptoms negative results of blood cultures should not contraindicate examination of the sinus.

The Ayer-Tobey test is a valuable diagnostic aid. The reliability of this test depends upon the experience and carefulness of the technician.

The records bear out definitely the common knowledge that the appearance of the sinus gives no indication of what may be within it. One has more reason, however, to suspect injury to the endothelial lining in cases in which there is a perisinus abscess.

Sinus thrombosis is not necessarily fatal if not treated surgically. Operation is always indicated, as the best means of aiding the patient to overcome the infection, affording drainage, thereby tending to limit the disease. Extirpation of the focus of disease is an impossibility; surgery merely aids the patient.

Postoperative sinus thrombosis occurs infrequently as compared with the manifest and latent types of thrombosis. Secondary infection of the wound of the neck occurs fairly commonly.

The percentage of fatalities in general may be considered to be between 20 and 30 per cent. In this report the patients who were hopelessly ill on admission were excluded. In the combined series there were 148 patients, of whom 32, or 21.7 per cent, died and 116, or 78.3 per cent, recovered. Excluding intracranial disease, sinus thrombosis must be considered to have a relatively high mortality rate.

TOBEY.

Epithelioma of the Middle Ear—Pathogenesis and Intracranial Complications (Complicazioni endocraniche e patogenesi degli epiteliomi dell'orecchio medio.)

Antognoli, G. C. (Rome), Riv. Oto.-Neuro.-Oft., 12:596 (July-Aug.), 1935.

Though only one ear case in 10,000 is malignant, and of such only one in six is an epithelioma of the middle ear, this author reports three cancers arising from the radical operative cavity, rapidly invading the petrosa and the cranial cavity and brain, developing from and showing throughout the tumor malpighian pavement epithelium.

The apparent cause seemed to be operative trauma superadded to the irritation of chronic otorrhea; also, in two cases, a luetic history. Histologic examination of exuberant granulations in radical cavities is recommended for persons over 30 who complain of severe headache during a prolonged convalescence.

FENTON.

Radical Mastoidectomy in Adults With Aural Tuberculosis and Active Pulmonary Tuberculosis.

Muskat, Irving (Chicago), Arch. of Otolaryng., 22:143 (Aug.), 1935.

Tuberculosis of the middle ear and mastoid, as seen in the adult with active pulmonary tuberculosis, because of its insidious, painless onset has not received the attention it merits.

Tuberculosis of the middle ear and mastoid presents an added focus to the pulmonary disease, interfering with the building up of bodily resistance which is essential to control of the disease. The focus in the mastoid, which is often extensive, can (unlike the pulmonary focus) be readily eradicated by the classic radical mastoidectomy performed with local anesthesia. Patients with pulmonary disease improve in health with the eradication of the focus in the mastoid.

TOBEY.

Influence of Large Doses of Vitamin D (Irradiated Ergosterin) on the Auditory Apparatus (Influenza delle dosi eccessive di vitamina D (ergosterina irradiata) sull ' apparato uditivo).

Scalzitti, M. (Roma), Valsalva, 11:85-93 (February), 1935.

Histologic researches were conducted on a litter of ten guinea pigs. They were fed on a normal diet to which 10 mgm. of pure irradiated ergosterin was added daily, in a 1 per cent oily solution.

The author observed on two of these ten guinea pigs, which were fed excessive doses of vitamin D, the following changes in the auditory apparatus, especially on the labyrinth:

1. Connective tissue metaplasia of the osseous capsule with thickening of the arterial walls.

2. Minute decalcified zones at the insertion of the ligament of the short process of the incus.

3. Zone of rarefaction at the articular surface of the stapes at the point where the cartilage is attached to the osseous tissue.

4. Marked dilatation of the modiolus causing its wall to become irregular and at many points very thin.

5. Increase in size of Corti's canal with dilatation of the afferent canaliculi.

6. Increase of loose connective tissue between the cochlear nerve fibers and the canal walls.

7. Marked decrease in number of cells of Corti's (spiral) ganglion which appear dilated while the connective tissue between these cells become increased in thickness.

8. The organ of Corti and Scarpa's ganglion are normal.

Four microphotographs accompany the article.

SCIARRETTA.

Unilateral Leontiasis With Auricular Manifestations (Osteopatia iperostotica leontiasica con manifestazioni auriculari).

Giuffrida, E. (Catania), Arch. Ital. di Otol., Rino. and Laring., 47:81-99 (February), 1935.

A description is given of a very rare case of bone hypertrophy, which began at the age of ten, limited to the left half of the face and cranium, and accompanied by marked loss of hearing. The patient is now 36 years old and the tumefaction has increased considerably since first observed.

The author considers this case among the chronic hypertrophic bone diseases which are represented by Recklinghausen's and Paget's diseases and leontiasis ossea.

He believes that while the present knowledge will permit the differentiation of Recklinghausen's disease from Paget's disease and leontiasis, it is not possible to differentiate the Paget's disease from leontiasis. The case reported, due to its characteristics, is to be considered to be either leontiasis or Paget's disease, as the two are also histologically identical. The hypoacusia is not of Paget's type, but it is grossly due to leontiasis lesion of the zygomatic process which blocks the left external auditory canal.

In regard to the etiopathogenesis of the case the author expresses the hypothesis of a trophoneurosis.

SCIARRETTA.

MISCELLANEOUS.

Delay in Learning to Talk (I ritardi del linguaggio).

Scuri, D. (Rome), Valsalva, 11:401 (July), 1935.

The author recommends that any child which by the third year fails to talk or makes only a few sounds must undergo careful hearing tests; if these prove negative such a child should be immediately drilled in phonetics, to avoid the dangers of mental retardation which might take place if such training be deferred to the sixth year. Obviously, deafened children will require lip reading and sound training by other accepted methods, from a similarly early period.

FENTON.

Modern Diagnostic Methods Used for Pneumoencephalography (I moderni metodi d'indagine radiologica del nevrasse. La pneumoencefalografia).

Bertolotti, M. (Milano), Riv. Oto-Neuro-Oftal. and Radio-Neuro. Chir., 11: 349-454 (July and August), 1934.

This article covers the entire issue of 114 pages. The subject is summarized to date and is profusely illustrated with clear and instructive roentgenograms and anatomic drawings.

The author presents first the anatomy of the ventricular and subarachnoid systems. Then he reviews the origin of the technic and the numerous variations employed by different authors; types of contrast-media and reasons for their selection.

He finally concludes with a discussion of the indications and contraindications for the employment of these diagnostic methods.

An extensive bibliography is also furnished.

SCIARRETTA.

Notices.

THE AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Cincinnati, Ohio, September 14, 1935. Fifty-seven candidates were examined, out of which number forty-two were certified and fifteen conditioned.

The Board will hold an examination in Kansas City, Mo., May 9, 1936, during the meeting of the American Medical Association, and in New York City, October, 1936, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology. Exact date has not been set as yet. Prospective applicants for certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, Neb., for application blanks.

W. P. WHERRY, M. D., Secretary-Treasurer.

H. P. MOSHER, M. D., President.

Among the speakers who will participate in the Fifth Annual Midwinter Clinic Course in Ophthalmology and Otolaryngology at Los Angeles, January 20 to January 31, 1936, will be Col. Robert E. Wright of the British Army Medical Corps at Madras, India; Dr. John F. Barnhill of Indianapolis; Dr. Edward Jackson of Denver; Dr. John Weeks of New York City; Dr. L. W. Dean of St. Louis; Dr. Isidore Friesner of New York; Dr. Thomas E. Carmody of Denver; Dr. Hans Barkan of San Francisco; Dr. Donald H. O'Rourke of Denver; Dr. Vern O. Knudsen of Los Angeles; Dr. Seeley G. Mudd of Pasadena, and Dr. Clifford B. Walker of Los Angeles.

The Registrar of the course is Dr. Pierre Viole, Treasurer, 1930 Wilshire Blvd., Los Angeles, Cal.

The Medical School of Washington University offers a one week's intensive course in Ophthalmology and Otolaryngology for qualified ophthalmologists and otolaryngologists. The course begins Monday, March 2, 1936, at 8:00 A. M.

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